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An Address.[1]

By K. C. T. RAWLE,

President of the New South Wales Branch of the British Medical Association.

I WISH to express my deep appreciation of the great honour which you have conferred on me by my appointment as president of the New South Wales Branch of the British Medical Association. I feel that the position is one of great responsibility, and it is my earnest hope that, with your confidence and loyal support, the work of this Association will continue to prosper in the coming year.

I would also pay tribute to our immediate past president, who has set a very high standard in carrying out his duties as president in a most efficient manner and has been responsible, with the support of Council and its officers, for much progress. The thorny problem of the coordination of the many conflicting types of contract industrial medical practice was solved under his expert chairmanship. Let us hope that one of this year's problems, a smooth transition from the old contract system of lodge practice to a more satisfactory form of service, will, with your help, be solved in an equally successful manner.

In accordance with custom, it is now my duty to deliver an address. In view of the fact that I have been actively engaged in general practice for many years, I feel that my comments should perhaps bear some relation to general practice as I know it, with particular reference to some of its current problems and suggestions for their solution.

[1] Delivered at the annual meeting of the New South Wales Branch of the British Medical Association on March 29, 1951.

In this, our Commonwealth Jubilee year, it is only natural to look back over the progress made during the past fifty years. This, however, has been very ably covered in our splendid Jubilee Number of THE MEDICAL JOURNAL OF AUSTRALIA of January 6, 1951, for the production of which I offer to the Editor my hearty congratulations. The authors have dealt fully with all aspects of practice, including general practice, both urban and country. I propose, therefore, to look at the problem from another angle, taking as my object lesson the present state of general practice in Great Britain. All that I have to say has been said before, and if you will bear with me for a short time, even if I stress the obvious, some practical solution may present itself to your mind which will help to raise the status of the general practitioner and of general practice in this country.

It is obvious to all who open their medical journals that all is not well with the medical profession in England. In practically every issue letters appear under the headings "G.P. at the Crossroads", "Difficulties of the G.P.", and so on. Articles published and the reports of proceedings of the Council of the Parent Body and its committees point in no uncertain way to the discontent that is abroad. While realizing that Australian conditions are not the same as those in England, I think that a little sober reflection will do no harm. A year or so ago the profession in Australia narrowly escaped from health legislation which would have brought us practically the same problems as confront the profession in England.

In the *World Medical Association Bulletin* of October, 1950, Dr. Charles Hill, Secretary of the British Medical Association and President of the World Medical Association, stated:

> The branch of practice which is perhaps causing most concern at the present time is general practice. Part of the anxiety of general practitioners in the National Health Service is caused by the Minister's delay in settling the profession's claim for higher remuneration on the basis of the Spens Report, and his failure to give an assurance that negotiations on general practitioners' remuneration will always be conducted through "Whitley" machinery (i.e. conferences of representatives of the "management" and "staff" sides). . . . Remuneration, however, is not the only factor which is worrying general practitioners. There is a feeling that general practice is losing its former prestige and status in the eyes of the profession as a whole and of the public.

In the Supplement to the *British Medical Journal* of August 12, 1950, appears an article by Dr. Frank Gray entitled "The Way Divides". He states:

> There is abundant evidence both that something is wrong and is felt to be wrong by a very large number of general practitioners. . . . Apart from the question of finance, which is receiving the fullest consideration elsewhere, the chief trouble appears to have been accentuated and displayed, but not originated, by the National Health Service Acts. It is the question of status.

A delegation of the American Medical Association which visited the United Kingdom in 1949 was particularly impressed by the plight of the general practitioner. They found him trying to cope with more patients than he could treat satisfactorily and consequently referring more and more patients to the hospitals, from which he himself was virtually excluded. The report continues:

> The general practitioner in Great Britain as we observed him is no longer a free, independent, professional person. He finds himself sharply circumscribed in function, in influence and in outlook. His work tends to become more superficial as he is increasingly concerned with clerical and technical trivialities. . . . The correction of present defects will depend on a reappraisal of the function of the family doctor and his re-establishment as the backbone of the medical service to the population at large.

These quotations paint a sorry picture. To one obvious comment, namely, "It can't happen here", let me answer —there is no room for complacency. The Act relating to medical benefits recently introduced by Senator McKenna is still on the Statute Book and has not yet been repealed. The reasons for this you know, but the fact remains and must be faced. While it may justly be claimed that the general practitioner in Australia has not reached and is not likely to reach the dreadful state mentioned in the above quotations, no harm—and indeed only good—can result from a similar reappraisal of the function of the family doctor in Australia. This, then, is my theme tonight.

In England, the profession itself has tackled the problem. The Council of the Parent Body appointed a committee under the chairmanship of Professor Sir Henry Cohen. The report of the committee is now available in a booklet known as "General Practice and the Training of the General Practitioner". It is not suggested that we should accept this report of the problems and suggested solutions as in any way applicable to our own Australian conditions. I do not propose to review the report, but commend it for careful study by all practising doctors, more especially those more closely connected with undergraduate and postgraduate teaching.

The Lancet of September 30, 1950, announces that the Northern Ireland committee of the Nuffield Provincial Hospitals Trust has decided to make an independent survey of the conditions of general practice in that region. The report continues:

> The main objects are to improve the status of the general practitioner and to find, if possible, how he can obtain reasonable time for leisure, contact with his colleagues, and postgraduate study. The assumption is that any improvement which can be made on these lines will be reflected in improved service to the patient.

What can we do to maintain and improve the status of general practice in this country?

The subject is a vast one and cannot be covered, even briefly, in the time at my disposal. To my mind, the obvious approach to this problem would be the appointment of a special committee to collect evidence by surveys, *questionnaires*, and direct interviews with the practitioners, and then to make recommendations; in other words, the reappraisal suggested by the American delegation.

Let us now consider a few of the points that will require careful thought.

Firstly, time: time to do one's work properly, time to take an adequate history, time to make a full physical examination where indicated, and last but not least time for leisure, that "leisure to grow wise" which, in well-chosen words, Dr. Alex Murphy, in his presidential address to the Australasian Medical Congress in Brisbane in May, 1950, referred to as the remedy—or palliative—to the march of cardio-vascular disease among the brain workers in their prime.

Secondly, a reduction of excessive clerical work. We must admit an irreducible minimum, but must resist by all means within our power the unnecessary increase in number and complexity of the various forms which plague us. Even the essential clerical work involved in one's own personal records needs constant review. In the *British Medical Journal* of September 16, 1950, appeared a stimulating article by Willis and Shaw entitled "Motion Study Investigation into the Clerical Work of a Doctor's Surgery". This dealt with practice in England. Would that someone with sufficient time and knowledge of modern business methods would work out and publish a time-saving, efficient and up-to-date system of filing, book-keeping and general management of the clerical side of a doctor's practice suitable for Australian conditions.

The next point which would require consideration would be the link between teaching hospitals and the general practitioner and the relation of this to the training of the undergraduate for subsequent general practice. One of the remedies suggested by the committee dealing with the general practice problem in England was more association of the general practitioner with hospitals. This is possibly very relevant in England, where the general practitioner has virtually been eliminated from the hospitals. This is not a major problem in Australia.

Let us consider some aspects of the teaching hospital in more detail. No matter how a hospital is organized, it remains the centre of treatment of established diseases, mostly serious and advanced. As such, the work carried out there represents curative work, or as it has been aptly named, "salvage". The hospital cannot cater for preventive medicine or the minor ailments of general practice. To describe one type of case in question, let me quote some words from an address by Dr. R. J. Perring, entitled "Family Doctor", published in *The Lancet* of December 24, 1949:

> Those of us working in general practice are only too familiar with the large proportion of patients coming to us complaining of a variety of symptoms which have apparently no physical basis and which must be emotionally mediated; womenfolk who after a few minutes' talk burst into tears, worried about—they know not what. The social havoc created by overcrowding, the difficulty of providing adequate meals, and the meagre family income in face of rising costs of living are evident enough; while home life with its jealousies and frustrations, working conditions with the inevitable boredom of constant repetition, and the perpetual striving for security or self-advancement produce problems more deep seated and therefore harder to solve. Many are insoluble.

This is not the type of case which is normally presented to the undergraduate student until such time as the second phase arises, that is, established disease, the duodenal ulcer, the thyreotoxicosis, the hypertension with its vascular accidents. The general practitioner, however, is in the picture long before this, when he is treating what has been referred to as the "trivial case". This includes the earliest stages of nearly all the stress diseases and other serious complaints, and being early, possibly curable. This is the stage at which the general practitioner is dispensing

preventive medicine and health education as distinct from the salvage work of the hospitals.

As medical students, we were all trained by consultants and specialists, men whom we admire and respect. We realize that their teaching was fundamentally sound, but it was on established disease. The conditions in the patients whom they treated were obviously conditions that mattered; the conditions which they disdained were of no importance. More junior honorary medical officers dealt with the out-patients, where earlier aspects of disease were seen and the sorting of patients was carried out. The only basically general practice of the hospital, that of the casualty department, was relegated to the junior resident medical officers. Thus was importance unwittingly given to the late stages of disease. The student, realizing these values, found that after graduation, when engaged in general practice, he was confronted with an aspect of disease which was the reverse of his early training and required a complete reorientation of his standards.

In this comment I do not in any way detract from the status or functions of the specialist, for the ever-increasing complexity of exact diagnosis and treatment has led to his evolution, but it has removed the art of medicine more and more from the intimacy of the bedside to the cold calculating atmosphere of the scientific laboratory. I would stress, however, that the aura of omniscience with which the specialist has been enveloped by the official mind and the public has led to a downward grading of the general practitioner. Yet it is the general practitioner alone who can view the whole picture and is finally responsible for adapting treatment to the need and ultimate well-being of the patient.

This gap in the training of the student for general practice is, I realize, being faced by all experienced teaching hospital administrators. The difficulties in the linking of general practitioners with the teaching hospitals have been discussed at length elsewhere. In Edinburgh, a scheme has been worked out and is worthy of very serious study in this country, with, of course, the necessary local adaptations. The excellent editorial in THE MEDICAL JOURNAL OF AUSTRALIA of March 24, 1951, covers this in detail. Some system can, I am sure, be eventually devised here.

A further point worthy of consideration by the suggested committee is means of obtaining the reestablishment of the right of the people to elect their own representatives to the boards of control of non-teaching hospitals, and the right of local practitioners to take their seats as full members of such boards, either as "staff" representatives or as elected members.

The committee might also profitably consider the question of the employment of recent graduates as assistants to established practitioners. There is on the Statute Book of New South Wales an Act requiring a graduate of medicine to occupy a post in an approved hospital for twelve months before being granted final and complete registration. Although this Act is in abeyance at present, I suggest consideration of the alternative of, say, six months as an assistant to an approved general practitioner. I feel that many recent graduates who intend to remain in general practice rather than tread the primrose path of specialism would avail themselves of this opportunity.

Time will not permit of discussion of many other points which will need to be considered. I can here mention only certain headings, such as post-graduate education of the practising general practitioner, a more satisfactory method of providing suitable "locums", methods of relieving the doctor's unfortunate wife of the many encumbrances and burdens of her association with medical practice, and finally, but by no means of least importance, the question of an increased remuneration for the general practitioner in keeping with modern values and the responsibilities accepted.

It may be argued that the Association already has the necessary machinery for permitting the general practitioner to become vocal, through resolutions passed by local associations and brought forward at the annual meeting of delegates. An analysis of the type of resolution which is usually brought forward at these meetings, however, to my mind points to the need for some further machinery capable of bringing forward and correlating information regarding general practice and of making recommendations.

I therefore conclude by suggesting that Council give serious consideration to the formation of a special committee with terms of reference as follows: "To review the present position in general practice, its difficulties and its trends, both generally and with reference to the British Medical Association report on 'General Practice and the Training of the General Practitioner', and to make recommendations."

I am confident that your cooperation with such a committee would be spontaneous and complete.

THE SURGICAL PATHOLOGY OF NON-SPECIFIC INFECTIONS OF THE NOSE AND ACCESSORY NASAL SINUSES.[1]

By GEORGE SWINBURNE.

From the Department of Pathology, University of Melbourne, and the Ear, Nose and Throat Department, Royal Melbourne Hospital, Melbourne.

AT the outset it may be stated that rhinitis and sinusitis, because of their close anatomical relationship, are closely allied from the clinical and histopathological points of view. Nearly always there is some degree of sinusitis associated with rhinitis, acute or chronic. For convenience of description they will be dealt with partly separately, but mainly together. The particular effects of acute and chronic inflammation on the different individual sinuses are largely beyond the scope of this paper.

ACUTE RHINITIS AND SINUSITIS.

Acute rhinitis and sinusitis means an acute inflammation of the mucous membrane lining the nasal cavity and to a greater or lesser extent the similar lining mucosa of the nasal accessory sinuses. Quite a number of diseases begin with such an inflammation, but the more important are (*a*) the common cold (synonym, acute coryza), (*b*) influenza, epidemic or pandemic, and (*c*) some of the exanthemata, especially measles.

Ætiology.

From the results of observation and both clinical and laboratory research it seems probable that the initial stage in this disease occurs when a filtrable virus gains a foothold in the mucous membrane of the nose, and so lowers its resistance that the common secondary invaders are able to multiply and attack it (Eggston and Wolff, 1947). It is probable that the characteristics of the mucous membrane are changed, and that there is an alteration in the mucin allowing the secondary invaders access.

The virus of the common cold has not yet been isolated, though the virus of influenza and that of some of the exanthemata have been (for example, rubella). The common secondary invaders include *Micrococcus catarrhalis*, streptococci, pneumococci, and *Hæmophilus influenzæ*.

Predisposing causes allowing the virus an opportunity to gain entry to the nose include the following: (i) Exposure to infection, especially close association with a person in the early stages of a common cold or influenza. Cold weather causes overcrowding indoors and throws people together often in ill-ventilated rooms. (ii) The ill-defined factors usually grouped under the heading of a lowered general resistance, due to overwork, loss of sleep, worry, excess alcohol intake or some constitutional diseases. (iii)

[1] The subject matter of this paper provided the material for one of a series of post-graduate lectures in pathology delivered under the auspices of the Melbourne Permanent Post-Graduate Committee to candidates for Part II of the Diploma of Laryngology and Otology in the University of Melbourne.

Injury to the nasal mucosa by exposure to cold or chilling. Swimming or diving for too long or in too cold water causes chilling; washing away or dilution of mucin is also a factor on these occasions. Local irritation from chemical fumes or dust may also injure the mucous membrane. (iv) Certain variations in nasal anatomy, which cause an obstruction and interfere with the normal physiology of the nose. Most of the above-mentioned factors are contributory in their effect; the effect of low temperatures on the inadequately protected body has an especially important place.

When the body has been chilled for a prolonged period contraction of the blood vessels of the skin and respiratory mucous membrane is produced. If this vascular contraction occurs for too long the consequent ischæmia interferes with the local secretory conditions on the surface of the mucous membrane.

Clinically it seems probable that in certain of the above-mentioned circumstances susceptible individuals may develop symptoms indistinguishable from the "common cold" due to the action of the secondary bacterial invaders without the preliminary stage due to virus infection.

It is also possible, at least in temperate and cold climates, that the virus of the common cold is freely present in the air of living rooms and meeting places in the more closely populated areas, and then only some trauma to the mucosa or a low blood antibody level is necessary to allow the virus to enter. The common cold does seem to be less prevalent in tropical climates. The secretion of the seromucous glands of the nose and accessory sinuses normally has a relatively high content of lysozyme, which is bactericidal for a variety of non-pathogenic bacteria, and which in high concentration has an inhibitory effect on pathogenic organisms (Topley and Wilson, 1946; Burnet, 1950; Burnet, Lush and Jackson, 1939). It is known that the serous fluid in which the cilia of the respiratory tract are bathed contains a proportion (1% to 5%) of the antibodies circulating in the blood, and this is important in the mechanism of immunity to influenza. It is possible that a similar mechanism plays some part in immunity to the common cold (Burnet, 1950).

In chronic inflammation of the nose and accessory sinuses some of the above-mentioned factors may operate and allow certain of these secondarily invading bacteria, which are constantly present in the mucous membrane in the chronic inflammations, to become more active and so produce an exacerbation from time to time. The patient then complains of "frequent colds in the head".

Histopathology.

In acute rhinitis the following stages are usually discernible in the pseudo-stratified ciliated columnar non-olfactory portion of the nasal mucous membrane: ischæmia, hyperæmia, exudation and extravasation of serum, which then becomes seropurulent, then frankly purulent; the latter becomes thicker, more tenacious and scantier and finally disappears with a return of the mucosa to normal.

The ischæmic stage is present when chilling is a factor, but not always when other factors operate and chilling does not.

There is an increased secretion of mucus from the seromucinous glands in the submucosa and from the ciliated columnar epithelium itself, as shown by the appearance of numerous goblet cells secreting mucus. At this stage the secretion is serous in character. The mucous membrane then becomes œdematous and a few polymorphonuclear leucocytes appear between the cells of the epithelium.

Some of the surface cells then become loose and are detached, and epithelial cells can be found in the nasal secretion during the first three days. The degree of epithelial cell loss varies with the severity of the infection, but it can progress down to the basement membrane. At the same time there is round-cell and polymorphonuclear infiltration of the subepithelial tissues, especially in the periglandular region; this is accompanied by an increased tissue fluid content, and the œdema so caused produces a reduction in the airway with obstruction to breathing. Vascular congestion also contributes to this.

An increasing number of polymorphonuclear cells can then be found infiltrating the mucosa and passing through to the surface, where the secretion becomes more purulent in character.

The process of repair of the ciliated columnar epithelium appears to take place even while there is still much secretion from the nose. After the acute stage has passed at about the fourth or fifth day the epithelial cells remaining begin to proliferate, forming a many-layered syncytial mass on the surface where the loss has been severe. These cells gradually take on a columnar form, and by the tenth day the epithelium has usually almost completely regenerated.

With each cold, especially if it is severe, theoretically some slight scarring should be left in the submucosa, but from clinical observation it would seem that if a common cold runs its normal course of ten to fourteen days and the epithelial loss is confined to the surface layers which regenerate rapidly, then the cellular infiltration of the submucosal layer completely resolves without new fibroblast formation. This must be so, because many people suffer from three to six colds per year for many years without any obvious change in the mucosa or submucosa in the interval state. It is only when for some reason the inflammation passes to the subacute and chronic stages that permanent submucosal changes occur, and these will be described in detail later.

Pathology in Relation to Symptoms.

The incubation period of a cold is one to three days. The congestive hyperæmic stage lasts from six to twenty-four hours. There are swelling and tension in the mucosa, causing a tight uncomfortable feeling in the nose and often in the post-nasal space. There is a general constitutional reaction, which varies in degree according to the severity of the infection. There may be lassitude, malaise, itching in the nose and post-nasal space, or a dry burning feeling in these parts.

Soon a serous or watery mucoid discharge appears, varying in amount but usually copious. Sneezing may occur frequently at this stage.

Fever may be present if the infection is virulent, and *herpes simplex* lesions often appear on the lips and around the nares and are referred to as "cold sores" by the laity (Burnet and Williams, 1939).

With the increase in polymorphonuclear leucocyte and epithelial cell content the discharge becomes increasingly purulent in character. Later as the secretion of mucus abates this purulent discharge becomes more viscous and tenacious and scantier, and finally it disappears with the return of the mucosa to normality.

Complications.

In a person whose nasal anatomy is normal and who is constitutionally in good health before the onset, the common cold usually runs its course without complications. When there is some anatomical abnormality which, combined with inflammatory mucosal swelling, interferes with the drainage from an accessory sinus by occlusion of its ostium, retention of purulent secretion in that sinus may occur. When there is a virulent infection *plus* a breakdown in the normal function of the Eustachian tube, an extension of infection to the middle-ear cleft may occur causing *otitis media.*

In swimming, as well as chilling, dilution of nasal mucus affects the defence mechanism by decreasing any bactericidal and bacteriostatic power this mucus may have (Eggston and Wolff, 1947; Topley and Wilson, 1946), and for this reason excess nasal douching as a form of treatment is probably inadvisable. Douching should be carried out only for a special purpose, such as helping to rid the nose of crusts, and then kept to a minimum. No pressure should be applied with a syringe, or infected fluid may be forced up the Eustachian tube, causing *otitis media.*

Diving into infected water in addition may force some of it either into a sinus or up one of the Eustachian tubes, and diving whilst one is suffering from a cold increases the risk of complicating *otitis media*.

Conjunctival infection may result either from retrograde spread via the naso-lachrymal apparatus or from direct infection, usually transferred by the fingers.

Distal complications occur by passage of the infecting organisms to the throat, larynx, trachea and bronchi.

Pathology in Relation to Treatment.

Because we have no counter to virus infection there is as yet no effective preventive agent against the common cold. The multiplicity of nostrums bears out this contention. However, one can try to control the spread of and damage done by the secondary invaders.

Chemotherapy is effective in those instances in which the organism or organisms are sensitive, but many people feel that one is not justified in using penicillin or other antibiotics or sulphonamide therapy for a common cold which is not severe or complicated, because of the risk of producing resistant strains. There is also a risk of drug sensitivity which may debar its use in a subsequent more serious disease.

Isotonic vasoconstrictor nasal drops and inhalations are used in an endeavour to reduce mucosal swelling about the ostia of the various accessory sinuses. Such mucosal swelling may reduce the size of these ostia to such a degree that normal physiological drainage by cilial activity is seriously interfered with. It may be physically impossible for some blobs of mucopus to pass through the reduced ostium of, for example, a maxillary antrum, even if the cilia could handle such a blob. Sometimes the swelling may be so great that complete occlusion occurs, mucopus collects under pressure and pain is suffered. The beneficial effect of maxillary antral lavage is solely due to the removal of quantities of mucopurulent secretion which collects in the dependent part of the cavity and clogs cilial activity.

The use of sulphonamide preparations which are not in solution but in suspension may act as an irritant, and even in solution, if it is too concentrated, they may interfere with cilial activity by virtue of hypertonicity.

Penicillin in too great concentration may do the same. It is doubtful whether instillation of penicillin, streptomycin, aureomycin or other antibiotic, say twice a week, without its parenteral administration at the same time could possibly be effective. It is not yet proven that antibiotics instilled intranasally reach the organisms in the *tunica propria* (though they may affect those in the secretion in the sinus), despite statements to this effect. Clinical observation would lead one to believe that good results achieved by antibiotics and sulphonamides are mainly, if not entirely, due to their parenteral administration; the good effect of lavage of the antrum, for example, is due to unclogging of the cilia, and when locally applied vasoconstrictors which leave the cilia acting have opened up the ostium, "Nature does the rest".

Another form of treatment which had a recent vogue was the parenteral administration of penicillin *plus* penicillin inhaled in the form of a fine mist by the "aerosol" method. This latter method is of proven value for certain lung conditions—for example, to reduce infection temporarily in bronchiectasis or in bronchitis, but it is difficult to assess just what local concentration is achieved in the accessory nasal sinuses, how beneficial is its effect on the infection in the mucosa, and whether most of the good effects are not caused by the penicillin arriving by the blood-stream, whether introduced by injection or by inhalation through the lung.

Lastly, admission of the patient to hospital with the hourly introduction of penicillin into a particular sinus (the maxillary or sphenoid sinuses are the best placed anatomically for such cannulization) through a cannula from early in the day until late at night will keep up a concentration for eighteen of the twenty-four hours. This last procedure involves some trouble and discomfort and is seldom necessary. The parenteral administration of penicillin should be used as well, and a test beforehand, of course, will have established the penicillin sensitivity of the infecting organism before such treatment is begun. Bacterial culture of secretion and sensitivity tests with penicillin, streptomycin, aureomycin or other antibiotic will determine which drug will be most effective, however used. The mucopurulent return from antral lavage is commonly used for culturing the organism (if this sinus is affected).

Growing reports in the literature of the value of short-wave diathermy (high-frequency currents) in the treatment of sinusitis should be viewed with some reserve. This treatment causes the production of heat in the body tissues lying in the field between the electrodes. It is used for therapeutic purposes, but the temperatures produced are not high enough to destroy tissues or impair their vitality. Zeiter (Glasser, 1944) makes the following observations in this connexion:

> With development of short wave currents, many claims have been made for special effectiveness of various wavelengths. Selective thermal action, specific biologic and bactericidal action and a more effective and less dangerous action in heating human tissues than conventional (or long wave) diathermy have been attributed to short wave diathermy. In a comprehensive review of these claims it was found that some investigators have not been able to substantiate the findings.

Zeiter gives references to the work of these investigators.

Experiments by Mortimer and Osborne (1935) show that a special selective thermal action is questionable.

It was also found that there was no apparent advantage in any particular wave-length (Mortimer and Beard, 1935).

Mortimer and Osborne could find no conclusive evidence in the literature, nor could they substantiate in their investigations the claim for specific biological action of high-frequency currents (short-wave diathermy). They believe the burden of proof still lies on those who claim any action other than that of heat. Experiments made to determine the specific action of short waves on bacteria have been performed in test tubes, and it remains to be demonstrated whether similar results can be secured with infection in the body.

With regard to sinusitis in particular, Zeiter makes the following further comments:

> Medical diathermy occasionally serves as a valuable adjunct to other routine forms of treatment in acute and chronic sinusitis. However, it is not the most important agent in treatment of inflammation of the nasal accessory sinuses. Diathermy may be most readily applied to the frontal and maxillary sinuses. It may relieve pain, but proper drainage should be established before high frequency currents are applied.

I agree with this latter statement; the production of further congestion in acute frontal sinusitis, for example, may be prejudicial to the patient's interests, because of the danger of occlusion of the fronto-nasal duct, the only means of egress for the excess secretion in the sinus consequent upon the acute inflammation.

In addition, Zeiter mentions as the first contraindication to the local application of high-frequency currents in general, acute inflammatory processes, such as acute non-draining cellulitis, acute infectious arthritis and acute pelvic infection.

Hollender (quoted by Zeiter; Glasser, 1944), in studies of the effect of short-wave diathermy in treatment of acute attacks of maxillary sinusitis, found that of 20 unselected patients six had to be given relief by irrigation, shrinkage and suction.

In a second series of 18 cases of acute maxillary sinusitis short-wave diathermy was the only form of treatment and gave the least satisfactory results of any treatment. In a third series short-wave diathermy was used in conjunction with shrinkage and irrigation, and in this group the response was the most favourable.

An uncontrolled series of X-ray films showing maxillary antra before and after treatment by short-wave diathermy every other day for six weeks with improvement does not necessarily mean that the short-wave diathermy effected

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the change. A control series of cases, in which treatment was by isotonic vasoconstrictor drops and inhalations in the correct strengths *plus* antral lavage at judicious intervals according to the response, has shown in our out-patients' department that this type of treatment produces better results. In acute rhinitis and sinusitis untreated for six weeks quite a proportion of patients can be shown to have completely resolved. This does not mean that short-wave diathermy has not a place in treatment, but it must be used with care and intelligence. In acute empyema of the maxillary antrum its use alone without measures to establish ciliary drainage could be futile if not dangerous, as its immediate effect is to increase the tissue fluid (including blood) content to the parts, hence increasing the congestion for the time being. Since the introduction of chemotherapy with sulphonamide drugs and the antibiotics into the treatment of sinusitis, the indications for short-wave therapy have almost disappeared. Its empirical use as a last resort in the treatment of chronic sinusitis gives poor results in my experience. This is also Zeiter's opinion.

FIGURE I.

Simple chronic rhinitis. Photomicrograph showing some hyperplasia of the epithelium, an increase in the number of permanently dilated blood vessels, œdema and thickening of the *tunica propria* and small round-cell infiltration, the whole producing the bogginess of the mucosa noted on clinical examination. (× 100.)

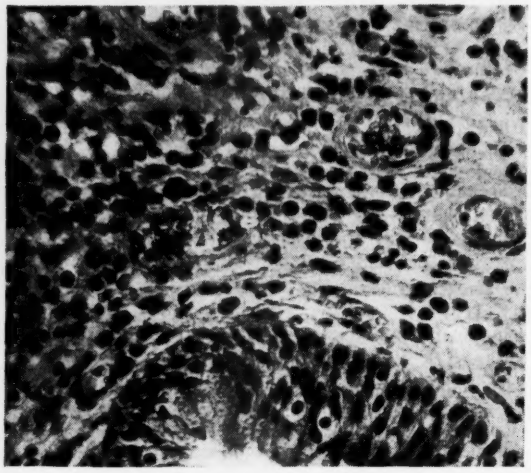

FIGURE II.

Simple chronic rhinitis. Photomicrograph showing some hyperplasia of the epithelium, an increase in the number of permanently dilated blood vessels, œdema and thickening of the *tunica propria* and small round-cell infiltration, the whole producing the bogginess of the mucosa noted on clinical examination. (× 350.)

CHRONIC RHINITIS AND SINUSITIS.

Like acute rhinitis and sinusitis, chronic rhinitis and sinusitis are closely associated. It is possible that their association is least in some forms of atrophic rhinitis.

Chronic rhinitis and sinusitis may be classified as follows: (*a*) simple chronic rhinitis (and sinusitis); (*b*) chronic hypertrophic rhinitis and sinusitis (synonym, chronic hyperplastic rhinitis); (*c*) chronic purulent rhinitis and sinusitis: (i) polypoid, (ii) sclerosing or fibrotic, (iii) glandular or adenomatous, (iv) papillary, (v) atrophic.

Simple Chronic Rhinitis.

Simple chronic rhinitis is also called chronic catarrhal rhinitis and chronic nasal catarrh (Figures I, II and III). In this condition there is a catarrhal inflammation of the nasal mucous membrane; the secretion is serous, not purulent. Swelling and bogginess of the epithelium are present, together with œdema and hyperplasia of the sub-epithelial tissues associated with a change in the amount and character of the secretion.

The condition occurs under the following conditions:

1. As a result of incomplete resolution of acute rhinitis, especially following frequent recurrent attacks, which may be associated with anatomical abnormalities such as gross irregular septal deviation producing continued nasal obstruction. Any of the predisposing causes mentioned in the section on acute rhinitis may contribute.

2. In latent chronic low-grade sinus infection—and the first type usually leads to the second type.

3. In children as a result of hypertrophied adenoid lymphoid tissue causing choanal obstruction and interference with nasal cilial drainage.

4. In certain persons who live in unhygienic surroundings and conditions, especially persons who follow certain occupations which expose their nasal mucosa to the irritating effects of chemical vapours such as ammonia, chlorine *et cetera*, or to an atmosphere containing fine particles of dust or debris (for example, millers, bakers, weavers, coal-miners, stonemasons, hay-cutters, grass-hay contractors). Many of the wood carvers and metal workers now have the benefit of mechanical dust extractors fitted to their benches.

5. In certain constitutional disturbances—for example, chronic renal, cardio-vascular or hepatic lesions, or gouty, rheumatic and diabetic diatheses. These are alleged to be predisposing factors.

Because of continued or repeated congestion of the local vascular apparatus, the dilatation associated with the continued hyperæmia becomes permanent to a greater or lesser degree. As well as this loss of contractility there is also a permanently increased permeability. This hyperæmia is generalized and not to be confused with that which normally occurs in the erectile tissue in response to changing temperature and certain other stimuli (hormonal), and which is controlled by a neuro-vascular mechanism (Swindle, 1935). The result is a chronic slight water-logging of the tissues and an increase in the fibroblastic content of the submucosa. Together these factors produce the swollen, hyperplastic, slightly indurated, boggy mucosa seen clinically.

The chronic distension of the vessels also affects the venous plexuses of the turbinates, adding to the permanent increase in size of these structures already contributed to by the submucosal changes described.

The epithelium itself is also affected by hyperplasia and thickening, the layers of cells are greatly increased in many places, and goblet cells are permanently present in varying numbers, their secretion contributing to the excess serous nasal discharge, the remainder coming from the submucosal sero-mucinous glands, which also show permanent hyperactivity.

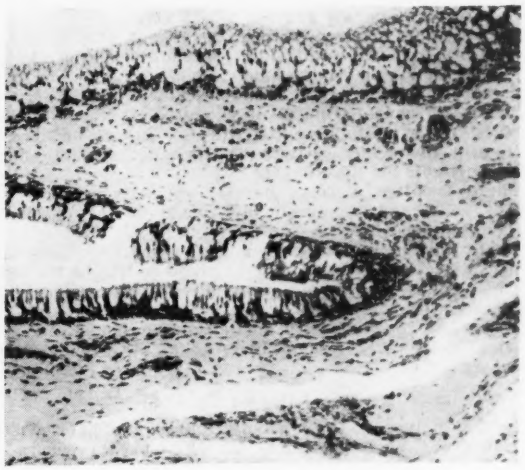

FIGURE III.

Simple chronic rhinitis. Photomicrograph showing a stage nearer to chronic hypertrophic rhinitis, an increase in the layers of cells (even when allowance is made for obliquity in the cut of the section), a great increase in the number of goblet cells, and early fibrosis. (× 150.)

The main symptoms are the obstruction to the nasal airway, usually intermittent and often alternating, and the excess serous discharge, which worries the patient both anteriorly and post-nasally.

This condition is especially worrying at night, the obstruction occurring usually on the dependent side. Voice changes occur because of the interference with nasal resonance. The sense of smell becomes impaired if the olfactory cleft is ill-ventilated. There is often a complaint of dulness and mental sluggishness. The patient suffers from attacks of acute rhinitis more frequently than the normal person.

There is often a sensation of tickling or itching in the nasal vestibule and anterior part of the septum, caused by the drying of secretion. This causes picking of the nose and abrasion and ulceration of the septal mucosa and in some cases ultimate perforation.

The colour of the swollen and boggy mucosa varies; it is pale in the anæmic and debilitated and in the aged, but redder than normal in the plethoric.

Diagnosis is clinched by touching with a probe and is confirmed by the application of a vasoconstrictor solution, when most of the swelling disappears. The indent of the probe disappears almost immediately.

In true hyperplastic rhinitis (see next section) the swelling is mainly permanent and there is a great increase in the fixed tissue elements of the submucosa, vasoconstrictor solutions cause very little contraction of the hypertrophied mucosal tissue, and the indent of a probe takes an appreciable time to disappear. It is a condition which can follow upon the simple chronic rhinitis under discussion.

Untreated simple chronic rhinitis may remain more or less stationary for years, but especially in the aged it can progress to an atrophic state. In the latter a continuous, annoying, watery discharge is present, but on examination there is no swelling of the mucosa and turbinates, but rather an atrophy of these structures, with a pale colour and an increase in the airway, not a decrease.

At any time, especially after a severe acute exacerbation, the condition may progress to chronic purulent rhinitis and sinusitis.

Treatment of simple chronic rhinitis, if early, may be successful, and consists in removal of the cause and restoration of the physiology of the nose to near normal if possible. The latter may involve constitutional treatment, local correction of anatomical abnormalities, or linear scarring of the submucosal tissue by diathermy cauterization to reduce the size of the turbinates and so restore the nasal airway. Removal of redundant tissue of the turbinates may rarely be necessary, but is used more often in the treatment of true hyperplastic rhinitis.

FIGURE IV.

Chronic hypertrophic rhinitis. Photomicrograph showing increased layers of cells and numbers of goblet cells in the epithelium, thickening of the walls of blood vessels, glandular hyperplasia and considerable permanent fibrosis. The condition of "mulberry hypertrophy" (*vide* text) develops only in certain locations, usually on the ends or lower borders of the inferior and middle turbinates, and sometimes on the *septum nasi* opposite the lower border of the middle turbinate. (× 150.)

Excessive and prolonged use of strong nasal vasoconstrictors, which is followed by reactionary vascular dilatation, tends to cause the condition or to prolong it if begun after its onset. Nasal douching with strong solutions has a similar effect.

Treatments which cause permanent damage to the ciliated columnar epithelium are inadvisable. Included in these are extensive chemical and galvanic cauterization of the surface of the turbinates and repeated use of zinc ionization.

Chronic Hypertrophic Rhinitis.

Chronic hypertrophic rhinitis, also called chronic hyperplastic rhinitis (Figure IV), is an end stage of simple chronic rhinitis, in which the long-continued inflammation has caused a big increase in the fixed tissue elements both in the submucosa and to a lesser extent in the epithelium itself. Also the walls of the chronically distended blood vessels become thickened. These two factors cause permanent loss of the normal contractility of the epithelial and subepithelial tissues. Diagnosis is helped by the effect of vasoconstrictors and probing, as described in the previous section. The final stage is papillary hypertrophy of the anterior and posterior ends of the inferior turbinates, and less often of the whole inferior border of that turbinate and occasionally of part of the inferior border of the middle turbinate and of the mucosa in the *septum nasi* opposite the anterior end of the middle and inferior turbinates. There is a mulberry-like appearance of the ends of the inferior turbinates. In the early stages the turbinate end is red and smooth, but

enlarged, then granular or lobulated; later it becomes pale and pedunculated as well as locally lobulated, the change in colour corresponding to an increased connective tissue content. The mulberry-like papillary hypertrophy appears because there is hyperplasia of the epithelium. It has not been satisfactorily explained why the hyperplasia is of such a regular pattern. The most likely explanation is anatomical. It is probable that the normal epithelium of these areas is bound down to the bony structure of the turbinate by fibrous tissue bands at regular intervals, as part of the mechanism for limiting the normal swelling due to the flow of the blood in the erectile tissue spaces which are present in the subepithelial tissue in the same areas.

The main symptom is nasal obstruction. This occurs especially at night, causing mouth breathing with dryness of the mouth and pharynx and secondary naso-oro-pharyngitis. There is also alteration in the resonance of the voice and in the sense of smell.

There is also a change in the character of the secretion and the amount; it becomes rather less in quantity than in simple chronic rhinitis, thicker and stickier or more tenacious. It has been described as like sago pudding. However, in some cases the discharge is still fairly profuse.

Often with the nasal obstruction some of the secretion is retained for too long in the nose and becomes a medium for the growth of saprophytic bacteria; it is then associated with an unpleasant odour.

The diagnosis is made as described for simple chronic rhinitis; the probe indents the swollen mucosa only slightly and the indentation fills in but slowly, whereas the mucosa pits easily and deeply in simple chronic rhinitis and the indentation fills in rapidly on removal of the probe.

Application of vasoconstrictor solutions does not cause much contraction of the mucosa and effects little improvement in the airway.

The presence of the mulberry type of hypertrophy of the turbinate ends or lower border confirms the diagnosis.

Treatment consists in removal of the excess tissue and the restoration of a reasonable nasal airway. Electro-coagulation diathermy used in a linear fashion may be a helpful additional measure when the hypertrophy of the tissue is not massive, but is not so useful as in simple chronic rhinitis.

Chronic Purulent Rhinitis and Sinusitis.

Chronic purulent rhinitis and sinusitis begins as acute rhinitis, and usually because complete resolution does not take place in one or more of the nasal accessory sinuses, subacute and finally chronic inflammation supervenes.

The cause is usually one or more of the factors mentioned in the section dealing with the ætiology of acute or simple chronic rhinitis.

As the condition persists and progresses, certain definite histo-pathological changes take place in the mucosa lining the particular sinus affected. This condition rarely affects the mucosal lining of the nasal cavities themselves, though a condition similar to that described under simple chronic rhinitis is usually present in the nose; chronic inflammation is present in the sinus or sinuses, and purulent secretion issuing from the ostia of these inflamed sinuses may be seen passing by the normal cilial routes over the surface of the turbinates and nasal cavities on its way to the naso-pharynx.

The reaction of the mucosa and submucosa to chronic inflammation as a result of bacterial infection varies; just why it varies in different subjects and in different circumstances has not been fully worked out. It may have some relation to the type of organism present, but no experimental evidence has as yet been put forward to suggest that this is the only factor. Infection with mouth organisms including anaerobes is known to produce a fairly definite type of reaction, but this may be described perhaps as being more acute than some others.

Eggston and Wolff (1947) have classified the types of histo-pathological reaction mainly on morphological grounds. The following is a modification of his classification.

Chronic Purulent Rhinitis and Sinusitis:

I. Polypoid (hypertrophic or hyperplastic sinusitis of Eggston).

II. Sclerosing or fibrotic.

III. Glandular or adenomatous.

IV. Papillary.

V. Atrophic.

Before I particularize in respect to the above groups certain general points may be mentioned.

In all types the epithelium in places may undergo metaplastic changes and the pseudo-stratified ciliated columnar character may become stratified columnar or even squamous. Loss of cilia in the pseudo-stratified area is common. Frequently purulent exudate is present on the surface of the epithelium. A varying degree of fibrosis occurs in the *tunica propria* of the mucosa, being most pronounced in the fibrotic type.

There is often a large number of actively secreting goblet cells, and round-cell infiltration of the epithelial layer often occurs.

There may be an increase of glands in the submucosa, and cystic changes may occur in these owing to blocking of their ducts with retention of the secretion. There is an increase in the vessels in the submucosa, and in many perivascular "cuffing" with small round cells occurs, and plasma cells are frequently present in considerable numbers.

The Polypoid Type.

1. The polypoid type (hypertrophic or hyperplastic sinusitis of Eggston, Figures V, VI and VII) occurs in chronic rhinitis and sinusitis of long standing. Although it probably always is the result of bacterial infection, it may be present without clinical evidence of suppuration. There is nearly always a sensitivity to bacterial protein on skin testing. Polypi are localized œdematous areas of hypertrophy of the mucous membrane; they may be sessile and at a later stage pedunculated. They are smooth and greyish-pink in colour, and appear as jelly-like or grape-like rounded masses covered by ciliated columnar epithelium unless they are exposed near the nasal vestibule, when the epithelium may change to the stratified squamous type. Their histological structure varies from the relatively dense fibrous type to the soft œdematous or even cystic type.

Eggston has made a particular study of the cause of nasal polyposis. After study of a great many specimens he maintains that repeated chronic inflammation with acute exacerbations causes fibrosis around the lymph vessels and the small veins, which have been the seat of long-standing perilymphangitis and periphlebitis. Finally, thrombosis of some of these small veins and lymphatics occurs. There is interference with the return flow of blood serum, causing passive congestion and œdema of the stroma, and if this occurs over a sufficiently large area polypus formation occurs.

This is a simple and plausible explanation, but close analysis raises doubt as to whether it is correct. There are two phases in the development of a polypus: the early and the late. Actually some polypi seen in the nose arise in the accessory sinuses, most commonly the maxillary antrum or one or several of the ethmoid sinuses. Others arise from the mucosa lining the middle or superior meati or from the borders of the middle turbinate, usually its anterior or less commonly its posterior end. The mechanism of origin—that is, the early phase of both groups (for example, one from the sinus mucosa and the other from the mucosa of the nasal cavity) is probably similar. Once the polypus has begun, physical and mechanical factors encourage its growth. This occurs certainly in some cases (for example, when the polypus has passed through the constricting ostium of the maxillary antrum or that of an ethmoid cell) and in all probability in others (for example, when the polypi cause nasal irritation, a feeling as if something is present which needs to be blown out, or frank obstruction to the nasal airway). Repeated attempts are made to expel or release the obstruction; these cause measurable alternating changes in intranasal pressure, which drag the pedunculated

polypus anteriorly or posteriorly. Ultimately the effect may be an increase in its length and size. The larger it is and the narrower its pedicle, the greater the interference with the venous and lymphatic return from it.

Eggston does not distinguish clearly enough between the early and the late stages of development of a polypus.

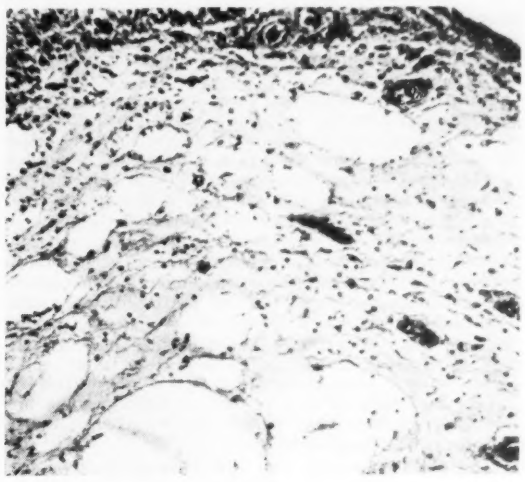

FIGURE V.

Polypoid type of chronic purulent sinusitis. Photomicrograph showing distended tissue spaces, to be distinguished from the true cystic type of polypus in that the spaces have no epithelial lining at any stage. (× 150.)

It is difficult on our present knowledge to disagree with him when he says that polypi are inflammatory in origin or follow changes consequent upon inflammation. Nevertheless, as was mentioned elsewhere in this paper, the

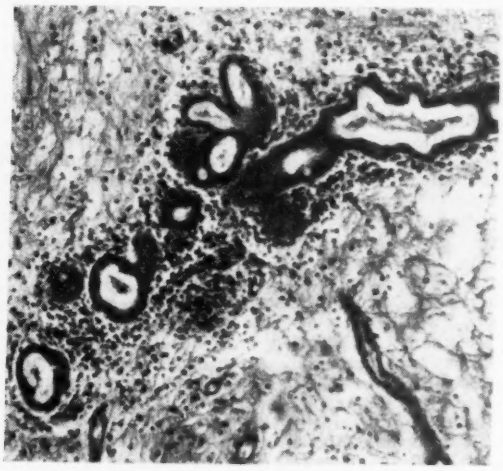

FIGURE VI.

Polypoid type of chronic purulent sinusitis. Photomicrograph showing cystic changes developing in seromucinous glands, and as well simple distended tissue spaces as depicted in Figure V. (× 150.)

question arises whether bacterial allergy is concerned, and it must be admitted that the actual mechanism underlying the origin of nasal polypi is not known.

To analyse critically Eggston and Wolff's words and their photomicrographs, one might remark that even though "repeated attacks of infection finally lead to a periphlebitis or a perilymphangitis", and presuming some fibrosis follows the small round-cell "cuffing" of the small veins and lymphatics, it does not necessarily follow that such fibrosis has a mechanical effect on these vessels. The control of vessels is not due to mechanical factors; it is probably largely due to chemical factors. Eggston and Wolff also state that "this mechanism of the production of polypoid sinusitis is similar to the production of hæmorrhoids"; but is this so? These authors also make the following statement:

> . . . that there is an obstruction to the return flow of the blood and fluids, with a congestion, and probably a thrombosis. The arterial supply remains intact. The fluids continue to enter the tissue, but drainage is interfered with. Consequently, the tissues become distended. With the distension, there is interference with oxidation of the tissues and nourishment. There follow changes in the physico-chemical reaction of the fluid medium such as ionic concentration, osmotic pressure and pH concentration resulting in an œdema and distension of the parts. The normally patent arteries continue to supply the fluid to the area, but little of it returns to the circulation, which is easily understood when the veins and lymphatics are obstructed. The tissue fluids that remain produce an œdematous hypertrophy of the involved area.

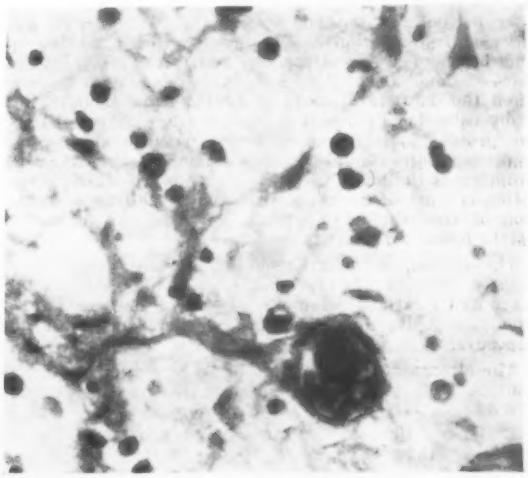

FIGURE VII.

Polypoid type of chronic purulent sinusitis. High-power photomicrograph of tissue at base of polypus, showing blood vessels containing blood cells, but no evidence of thrombosis. (× 650.)

This explanation is open to considerable doubt in my mind. In the study of my own microscopic sections thrombosis of vessels has not been present to any extent, if at all (*vide* Figure VII). Eggston and Wolff's examples of thrombosed vessels are not convincing. It is a pity some higher magnifications of these vessels have not been given.

Even if these examples were genuine thromboses, many non-thrombosed collaterals are evident in the sections. The analogy of the production of hæmorrhoids is hardly correct. When thrombosis occurs in an external hæmorrhoid the whole hæmorrhoid is affected, and if the clot is not evacuated it becomes fibrosed and is reduced to a fibrous tag. I have yet to see a thrombosed polypus. Furthermore, if thrombosis of a main return vessel (not demonstrated by Eggston and Wolff) did occur in a polypus and the arterial supply remained intact, and if the polypus became distended as they state that it does, one would see polypi as such clinically; but one does not. They are not tense—they are soft and rather flabby things, except the older slow-growing fibrous polypi, which are a little firmer but not tense. If the said conditions

held, the arterial inflow would quickly push the pressure in the polypus up to something of the order of 30 to 40 millimetres of mercury. The normal pressure in the veins of the body (and especially the head, which has an additional head of pressure due to gravity) above the right atrium of the heart is relatively low. In the nose the venous pressure may not be more than five millimetres of mercury (*plus* atmospheric pressure of course).

Eggston and Wolff make the fundamental mistake of not distinguishing between the late and early stages of hæmorrhoids and of applying facts obtained from observation of the late stages of hæmorrhoids to explain the early stages of the development of nasal polypi. The progress of the later stages in the development of hæmorrhoids depends largely on mechanical factors, whereas in the early stages the factors are probably mainly chemical and hormonal. Thrombosis in hæmorrhoids is a late complication.

It is also difficult to accept the hypothesis advanced by Eggston and Wolff that the œdema of the soft tissues in a polypus presses on and obstructs the duct of a mucinous gland and causes it to become cystic (their Figures 359 A and B; *vide* the description of a cystic polypus below).

Eggston and Wolff explain as follows the well-known rarefaction of bone in the walls of sinuses which occurs in long-standing polyposis. If the œdematous state is present for a period, a water-logged condition of the bone follows, the chemical reaction of the tissues alters, and the bone cells begin to act as osteoclasts and bone absorption takes place. Bone absorption may take place by halisteresis when the decalcification is generalized and usually rapid, or by osteoclasis when it is localized and slower. This is the process loosely spoken of as pressure absorption of bone especially found in the ethmoid labyrinth. This thinning of the bone has to be remembered when this condition is treated surgically. It actually facilitates exenteration of the ethmoid labyrinth, but care has to be taken not to break through its boundaries.

The epithelium covering nasal and sinus polypi may be ciliated columnar in type, but often metaplasia has taken place and stratified columnar, or cuboid, or even stratified squamous epithelium is found if there has been much exposure.

The stroma of polypi varies in its structure and cellular content within wide limits, according to the degree of (*i*) œdema, (*ii*) fibrosis and (*iii*) degenerative changes.

Distended tissue spaces are found which are not true cysts, as they have no epithelial lining. True cystic changes do occur in the glands of the stroma in polypoid conditions, and it is interesting to note that Eggston and Wolff maintain that these changes are secondary to pressure on the ducts from without—that is, from fibrosis or even œdema in the stroma—and not primarily due to occlusion of a duct previous to any other changes in the connective tissue, as occurs ordinarily in a mucous cyst (Swinburne, 1939). As has been stated, this is open to doubt.

Leucocytes may migrate into these cysts and desquamated epithelial cells may collect there with the mucus. If these cysts become infected, the content becomes purulent and accounts for at least some of the intramucosal abscesses found at operation. The cellular content of the stroma also varies according to the type; for example, some fibroid types have a relatively smaller cellular reaction. In others lymphocytes, plasma cells, histiocytes or large mononuclear leucocytes are found, and in the presence of more acute inflammation a varying number of different types of leucocyte.

Eggston and Wolff hold that the presence of eosinophile cells, if they are in great numbers, is suggestive, but does not necessarily mean that an allergic condition is present. When chronic inflammation in the sinuses has reached the recessive stage, and if regeneration has begun, there will usually be an increase in the number of eosinophile cells.

Myxomatous change is common in the œdematous connective tissue stroma of large polypi.

According to their histological structure polypi can be arbitrarily divided into several types: the œdematous, the fibrous, the angiectatic, the glandular and the cystic.

The œdematous polypus is the commonest type. The various types of metaplastic changes in the epithelium described above occur, but usually they retain a considerable area of pseudostratified ciliated columnar epithelium. Goblet cells are scanty, and degenerative changes have occurred in the *membrana propria*; often there is hyaline swelling; another common finding is disappearance of the elastic fibres, the epithelium then resting on a thin film of collagenous fibres.

The *tunica propria* has a loose network of connective tissue fibres with scanty spindle or star-shaped fibroblasts. There are a few elastic fibres. The spaces formed by these fibres and cells are filled with fluid, which is an albuminous transudate containing mucin. Round-cell infiltration is mainly around the blood vessels and glands beneath and close to the epithelium, but sometimes in isolated foci elsewhere.

The cells are mainly lymphocytes, with plasma cells and a few mononuclear cells; polymorphonuclear cells are often mainly eosinophile cells, with a few neutrophile cells unless ulceration has occurred.

The mucous glands lie near the surface and are often atrophied in this condition; this is said to be due to the pressure of the œdematous fluid, but is more likely the result of interference with the blood supply. The blood vessels are few and thin-walled and relatively small in calibre except in the pedicle.

In the fibrous polypus very few cilia are left; most of the surface is covered by stratified squamous epithelium with islands of stratified columnar type. The basement membrane, though thin, is pronounced and dense. The *tunica propria* is much less œdematous than in the other types; more fibroblasts and collagen and elastic fibres are present. There is probably relatively less cellular infiltration, and the cells are lymphocytes and plasma cells mainly, with a few polymorphonuclear cells. There are very few glands, and the few blood vessels have comparatively thick walls, partly due to increase of fibrous tissue, part of the general inflammatory reaction. In some cases there is an increase in the muscle content of the wall, and in others endothelial hyperplasia is present.

In the glandular type of polypus there is a relative increase in the amount of glandular tissue, mainly in the submucosal layer, and the glands are actively secreting; the lumina of the acini are filled with secretion, the epithelium of the ducts and acini is many-layered and the cells are larger than normal, numerous goblet cells being present. The glands nearer the centre of the polypus are smaller and less active and often show degenerative changes, in that the cells are losing their outline and epithelial debris is mixed with secretion in the lumen.

The cystic polypus really develops from a glandular polypus. The hypotheses are as follows: the epithelial debris mentioned above obstructs a duct (the most likely hypothesis—Swinburne, 1939); or, as some hold (Lederer, 1944), sometimes the œdematous tissues press on the duct; or, as others contend (Eggston and Wolff, 1947), fibrosis from long-standing inflammation constricts the duct; or a combination of these factors operates. The end result is that secretion and further desquamated epithelium accumulates in the alveoli, finally distending the whole collection of alveoli drained by the obstructed duct into a large cystic cavity lined by flattened epithelium or later by fibrous tissue only.

The content of desquamated degenerated epithelium and fatty debris may become infected and purulent, or hyalinization and even calcification may occur in the walls of the cyst. A similar histological picture occurs in the stroma of an infected air sinus in glandular sinusitis, and not necessarily with polyposis at all.

One other type may be mentioned, which has been called the angiectatic polypus because it has many blood vessels in the stroma. These have a distinct cellular wall and are dilated venous sinuses; they give a histological appearance resembling that of angioma.

Several of the above-mentioned types of histological appearance are often found in one large polypus or in the lining of one accessory sinus.

Hyperplastic or polypoid sinusitis causes nasal obstruction, especially if the polyposis is gross; it also causes anosmia (a mechanical effect mainly) and often, but not always, sneezing and a watery nasal discharge. Commonly, bronchial asthma is associated. The polypi may ultimately protrude into the naso-pharynx or become visible at the anterior nares.

The response to vasoconstrictors varies with the vascularity of the polypi. Treatment is by surgical removal of polypi in the nose to restore an airway. In some of these cases there is an associated allergy, but in others polyposis is associated with infection, and skin tests to all the common allergens produce negative results except to the protein of bacteria. The question of bacterial allergy must not be lost sight of in this connexion, especially as so far no convincing hypothesis explaining the origin of polyposis has yet been published. At present the tendency in treatment is to remove polypi only as is found necessary, to treat any allergic condition, and to avoid radical sinus surgery if possible (see the section on allergic rhinitis). Those cases associated in which a frank infection alone is present must be treated by adequate drainage. Those cases in which a mixed infective and allergic basis is present should be warily approached, and if surgical treatment other than simple removal of the polypi is decided upon, half measures are worse than none and the associated allergic condition must be treated.

Sclerosing or Fibrotic Sinusitis.

In the fibrotic form of purulent rhinitis and sinusitis (Figures VIII and IX)—and the fibrotic form is found more in the sinuses than in the nasal cavities—areas of stratified columnar and to a lesser extent stratified squamous epithelium are found interspersed with others in which the ciliated columnar characteristic is relatively unimpaired.

Considerable purulent exudate lies on the epithelial surface. The *tunica propria* is uniformly thickened, and throughout it is both a small-celled and a large round-celled and polymorphonuclear infiltration; there is an increase in the fixed tissue elements. The glands are normal or less than normal in number and activity.

The blood vessels have moderately thickened walls and, despite the considerably thickened *tunica propria*, are not conspicuously increased in number.

Despite these evidences of chronic inflammation there is not the offensive odour of atrophic rhinitis, and the hyperplastic changes in the mucosa or glandular elements seen in some forms are absent. This form may be described as simple chronic purulent rhinitis.

Glandular or Adenomatous Sinusitis.

In glandular or adenomatous sinusitis (Figure X) there occurs gross hyperplasia of the sero-mucinous glands, to such an extent that the microscopic picture suggests an adenomatous nature at first glance. The glandular tissue is increased in bulk and is hyperactive, the epithelium of the ducts and acini is many-layered, active cells being larger than normal and the number of goblet cells being increased. The lumina of the acini are filled with secretion.

At a later stage changes occur similar to those described in the section dealing with glandular and cystic nasal polypi. For one of a number of possible reasons discussed in that section the ducts of some of the groups of acini of the glands become obstructed. This happens because of the collection of epithelial debris in the duct lumen (the most likely reason—Swinburne, 1939), or as a result of œdema of tissues causing pressure on the duct externally, or because of fibrosis from long-standing inflammation around the duct constricting it, or from a combination of these factors; the end-result is a collection of cystic dilatations in the glandular tissue in which infection frequently occurs. Degenerated epithelial cells, mucus, leucocytes (active and degenerating) and bacteria are found in the dilated cystic spaces. The epithelium atrophies and the now pus-containing cysts are lined by fibrous tissue, with perhaps areas where a few flattened epithelial cells remain. These cysts are found at operation as intraepithelial abscesses. In occasional cases, usually in a maxillary sinus, a multilocular condition may be produced.

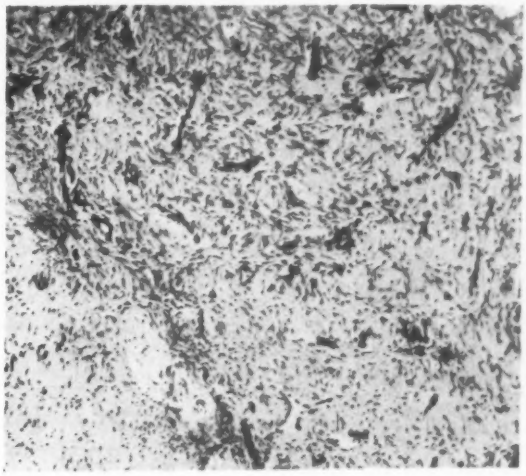

FIGURE VIII.

Fibrotic or sclerosing type of chronic purulent sinusitis. Photomicrograph showing a great increase in the fixed tissue elements. (× 100.)

Papillary Hypertrophic Sinusitis.

Papillary hypertrophic sinusitis is a rare condition mentioned by Eggston and Wolff (1947) and first described by Heymann (1900), when he drew attention to what he called a "non-malignant epithelioma" of the accessory nasal

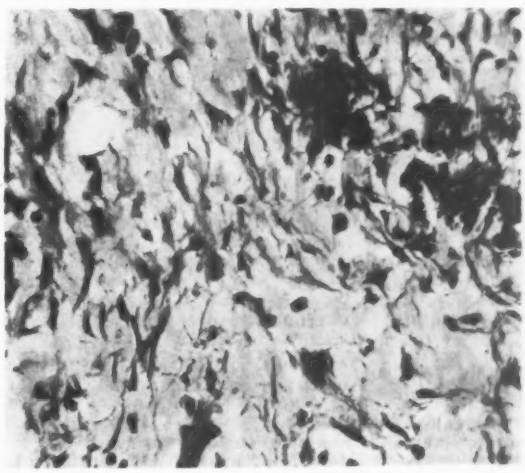

FIGURE IX:

Fibrotic or sclerosing type of chronic purulent sinusitis. Photomicrograph showing a great increase in the fixed elements. (× 300.)

sinuses. It usually occurs in the maxillary antrum, less often in another sinus, and the antrum may be filled with a papillary cauliflower-like growth. Histological examination reveals hyperplasia of the surface epithelium, often with metaplasia of the ciliated columnar type into the

squamous type and a heaping up of the cells into a typical papillary structure. In many places considerable cellular proliferation takes place and masses of these cells may penetrate deeply into the *tunica propria*; occasional mitotic figures may be seen, but careful examination will reveal an intact basal layer. Small abscesses are usually scattered throughout the mucosa.

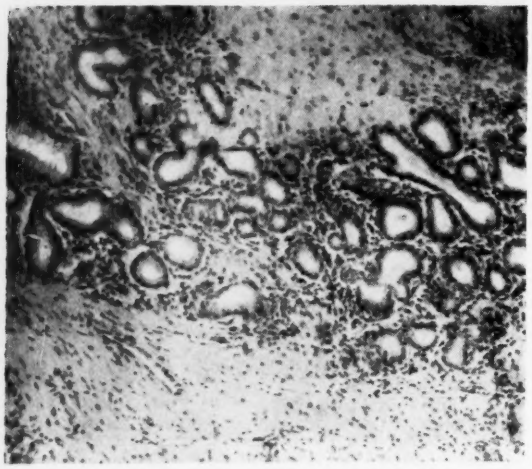

FIGURE X.

Glandular or adenomatous type of chronic purulent sinusitis. Photomicrograph showing gross hyperplasia of the sero-mucinous glands. (× 150.)

If the condition is suspected, the examination of several sections may be necessary to confirm the innocence of the condition and its inflammatory nature. An incorrect diagnosis of malignant disease has been made. Eggston thinks that a filtrable virus may cause this condition, but this appears to be supposition only.

Eggston has pointed out that the follicular sinusitis described by Ash (1939), in which lymphocytes are grouped together in lymph follicles or nodes, may occur in polypoid and papillary sinusitis, and is primarily a manifestation of chronic inflammation and not a separate type of sinusitis from the histological point of view.

Atrophic Rhinitis and Sinusitis.

Atrophic rhinitis and sinusitis (ozena, *rhinitis sicca*) is an atrophic condition affecting the mucous membrane and the underlying bone of the nasal cavities. It occurs to a lesser extent in the accessory sinuses. It is associated with the formation of offensively smelling crusts, removal of which reveals an abnormally large airway. It frequently affects the naso-pharynx, less often the pharynx and larynx, and rarely the trachea and bronchi.

From the point of view of ætiology, atrophic rhinitis can be divided into three groups, as follows. (i) That following trauma. This condition sometimes follows excessively radical sinus surgery, in which the anterior and posterior ethmoidal and spheno-palatine arteries have been injured or interfered with. The outlook for cure is bad. (ii) That associated with chronic infective sinusitis possibly due to certain specific organisms, or following a particularly acute exanthem originally. The former type is usually discovered early and may be favourably influenced if the infection in the air sinus is successfully dealt with; but in that following a severe exanthem permanent damage has usually been done to the mucosa. (iii) That of unknown origin, or the idiopathic type, the treatment of which is palliative only.

The hypotheses put forward to explain the ætiology of the idiopathic type are many, but none are proven, and the results of treatment based upon them have been uniformly unsuccessful. These include the hypotheses that the condition is a constitutional disturbance having an hereditary basis; that it is endocrine, trophoneurotic and anatomical in origin; that it is a terminal stage of chronic hypertrophic rhinitis, the fibrosis finally interfering with the blood supply. However, this cannot always be so, as the atrophic stage is not always preceded by the hypertrophic stage.

Syphilis has been held to cause some cases of atrophic rhinitis; but though gummata of the nasal cavities may produce a condition very like atrophic rhinitis, after antispecific treatment very few of these patients are left with the condition.

With regard to the anatomical factor, it is true that atrophic rhinitis is not uncommonly found in subjects who have certain bony abnormalities of the upper bones of the face and in the shape of the skull. They have a broad, flat face, large nasal cavities with small turbinate bones, and a sunken nasal bridge not unlike that of a congenital syphilitic, but with negative serological findings for that disease.

Some observers maintain that these abnormalities are part of the local atrophic change, and others that they are present before the atrophic mucosal changes and contribute to their production.

The early stages of atrophic rhinitis are not always easy to recognize, and clinically it often resembles advanced chronic hypertrophic rhinitis. There is a tough, firm look about the mucous membrane and a loss of resilience to touch with the probe. Sometimes this fibrotic type of mucous membrane, which is covered with stratified squamous epithelium, is found interspersed among patches of the hypertrophic type covered with stratified columnar and pseudostratified ciliated columnar epithelium. There is a progressive decrease in the younger connective tissue in the submucosa, with a diminution in the number of blood vessels and of glands. At a later stage the mucous membrane is frankly fibrous, and atrophic bony changes have occurred with decrease in size of the bony inferior and middle turbinates. The remaining arterioles are affected by endarteritis and periarteritis. Apparently no ulceration occurs except that caused as a result of trauma sustained in the removal of the crusts.

Eggston and Wolff hold that the sclerotic changes in the arterioles are the fundamental cause of the other atrophic changes, including a diminution in the number of capillaries and venous sinuses; the poor blood supply produces fibrosis in the stroma, which as it matures becomes more compact; the glands atrophy, the surface epithelium becomes more and more stratified squamous in type, and finally the secretory function of the mucosa with its important bactericidal powers is impaired or lost.

Because the ordinary cilial onward sweep of mucoid secretion does not occur, secretions accumulate and dry out and crusts form. These become the seat of infection with organisms, and it is infection with saprophytes such as the *Coccobacillus fœtidus ozænæ vel nasi* of Perez and Hofer (Topley and Wilson, 1946) and the *Bacillus mucosus ozænæ* of Loewenberg and Abel (Topley and Wilson, 1946) that causes the objectionable fœtor nearly always accompanying the disease. In 1925 Blane and Pangalos (Topley and Wilson, 1946) isolated from the nose of patients suffering from ozena a short, actively motile Gram-negative bacillus which they called *Bacillus ozogenes*. The *Bacillus mucosus ozænæ* belongs to the Friedländer group. *Proteus vulgaris*, *Bacillus Friedländer* and a pseudo-diphtheritic bacillus have been grown on culture, as well as staphylococci and streptococci. These organisms have been regarded as secondary invaders rather than as causative agents.

The main symptoms are the formation of crusts in the nose, which cause obstruction and are often difficult to remove without special aid, and the fœtid odour associated with them. The patient's sense of smell is grossly impaired or lost, and so he is spared the annoyance of the odour, though he is acutely conscious of its effect on his fellows.

Before actually forming crusts, the secretion is thick and viscid, and is inclined to adhere tenaciously to the mucosa.

The crusts are usually greenish-yellow, but this colour varies somewhat according to the predominant organism present. As the condition progresses and a larger mucosal area is involved, the crusts become larger and harder to expel.

The naso-pharynx is nearly always involved as well, and the crusts there are especially irritating to the patient, causing continuous hawking in an attempt to get rid of them. The mucosa of the pharyngeal wall becomes dry and glazed as the condition progresses downward.

When the larynx is involved (*laryngitis sicca*) hoarseness and a hacking cough develop.

When the trachea is involved, sometimes the patient has difficulty in actually ejecting the crust through the laryngeal aperture, and the subglottically impacted crust tends to add to its own size; finally, because of severe dyspnœa, laryngoscopic and bronchoscopic removal has to be carried out.

Rarely the condition may extend to the middle ear via the Eustachian tube and cause serious impairment of hearing.

Headache of a generalized type is often associated, though actual nasal or sinus pain is uncommon. Mental depression may be partly due to the organic lesion, but is mainly due to the patient's sensitivity with regard to the effect upon his fellows of the odour emanating from his nose.

The diagnosis depends upon the finding of the crusts and viscid, tenacious secretion giving off the characteristic foul odour, together with the atrophy of the nasal mucous membrane and turbinate bones and a great increase in the airway.

In the differential diagnosis, syphilis, foreign bodies, including rhinoliths, and chronic sinusitis (treatment of which may lead to cure in a small group of cases) must be considered. Some authorities mention lupus and scleroma, but it is doubtful if these would be mistaken.

Externally the saddle-shaped nose in some cases of atrophic rhinitis may lead to a suspicion of syphilis. Apart from the presence of other signs of syphilis elsewhere in the body, the history and the results of serological tests, the intranasal lesion is usually more localized in syphilis, and although it involves bone there is not the widespread atrophy of the mucous membrane and turbinates.

Treatment, except in the occasional early case associated with a chronic sinusitis, is palliative only. Permanent damage to the specialized nasal mucosa, if extensive, is irreparable. Efforts are directed toward loosening the crusts to make them easier to remove. Vasoconstrictor drugs are contraindicated, and solutions of vasodilator drugs can be tried; but any hope of success would be confined to early cases. Tampons of glucose (20%) in glycerine loosen the crusts; gentle lavage with an isotonic solution of salt and sodium bicarbonate then removes them.

Mechanical removal of some of the more resistant crusts may be occasionally necessary. Operative treatment designed to reduce the size of the nasal airway has not been particularly successful.

Allergic Rhinitis and Sinusitis and Vasomotor Rhinitis.

Allergy: Principles and Theory.

A number of clinical manifestations are today recognized as being due to allergy, or a sensitivity of certain mesenchymal cells of the human body to certain proteins. The clinical signs and symptoms are the result of the reaction produced in these sensitized cells on fresh contact with this particular protein. Chief of these clinical manifestations are bronchial asthma, allergic rhinitis both seasonal (hay fever type) and perennial, certain forms of dermatitis, serum sickness and serum shock, urticaria and angioœdema, which may have cutaneous, cerebral or visceral manifestations. (For the pathological appearances in allergic rhinitis and sinusitis see Figures XI and XII.)

This same sensitivity concept has been lately increasingly used to explain certain stages or lesions in many diseases, examples being the arthritis of rheumatic fever and the exanthem of scarlet fever and certain lesions of tuberculosis, the sensitivity being to a protein in the particular organism causing the disease.

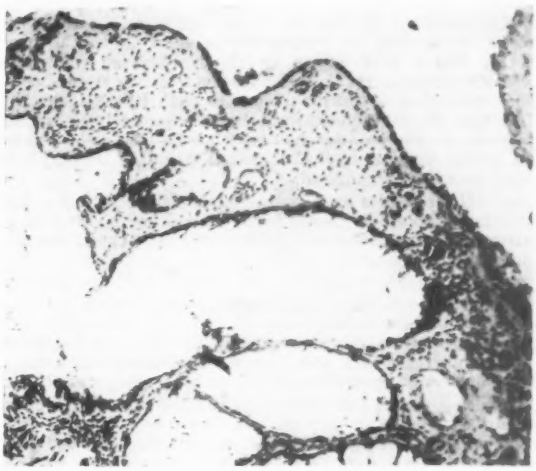

FIGURE XI.

Allergic rhinitis and sinusitis. Photomicrograph showing œdema and eosinophilic cell infiltration. (× 100.)

Blackley in 1873 showed that pollen caused hay fever; he performed skin and eye tests, and recorded local weal and systemic reactions in subjects with hay fever, and negative responses in normal controls. His excellent work was unnoticed.

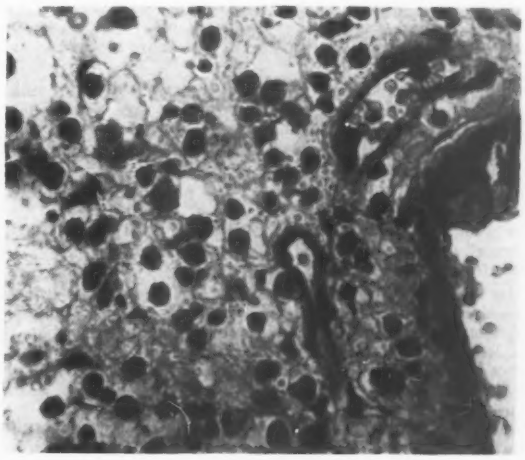

FIGURE XII.

Allergic rhinitis and sinusitis. Photomicrograph showing œdema and eosinophilic cell infiltration. (× 550.)

In 1890 von Behring discovered diphtheria antitoxin. Antitoxic horse serum was used to combat diphtheria, and after its use serum disease and serum shock in man and anaphylactic shock in animals were reported and then shown to be due to the serum proteins and not to the antitoxin.

In 1904 Theobald Smith, while working with diphtheria antisera, not toxins, noted the sudden death of guinea-pigs in convulsions on reinjection of the serum ten days after

a first dose which had produced no ill effect. A newly coined word, "anaphylaxis" (Richet, 1902), was applied to this phenomenon.

The present hypothesis, which was first postulated over twenty-five years ago, is that the primary and harmless parenteral introduction of foreign protein stimulates cells of the recipient to the production of specific antibodies, some cellular and the remainder circulating in the blood serum, which acts as an overflow reservoir for excess antibody (Cooke, 1947). Anaphylactic shock occurs when the specific cellular antibody unites with its antigen. But it is not known why certain cells respond with specific antibody production, nor just which cells they are; nor can the actual sudden explosive reaction called anaphylactic shock be wholly explained.

In 1921 Prausnitz and Kustner demonstrated that a skin-sensitizing factor is transferable by the serum of allergic patients to non-sensitive test subjects (the Prausnitz-Kustner reaction).

Then, in 1935, Cooke *et alii*, while investigating the reason for the clinical benefits occurring during the treatment of hay fever with pollen extracts, found that the protective mechanism was a specific blocking or inhibiting antibody (precipitin), which could be demonstrated in the blood serum of treated patients.

These represent the main milestones in the development of our knowledge of allergy; but there still remain many gaps, and for detailed discussion of these the reader is referred to text-books on this new and separate specialty.

Cooke and Vanderveer (1916) have estimated that approximately 7% of the population develop one of the regularly accepted group of allergies—for example, allergic coryza, seasonal and perennial asthma, the urticaria-angioœdema group and the allergic dermatoses.

Allergy, which means altered reactivity, was a word coined and first used by von Pirquet in 1906 to describe the alterations of reaction which he found to occur in man in response to second or later injections of smallpox vaccine and of horse serum. It is a word which has come into general use to cover a special group of diseases and symptom complexes caused by the interrelations of the various antigen-antibody reactions of man and of animals, and of which even the author of the word does not know the meaning.

The mere existence of an allergic state does not of course constitute disease. The disease is a result of the biochemical products or biophysical forces released when antigen and sensitized tissue cells unite (Cooke, 1947).

It is not known what it is that determines for the particular individual the tissue or part (shock organ) to be involved, or the pathological or immunological form the reaction will take. However, any organ or mesenchymal cell can apparently be so affected, so the clinical manifestations are many and varied.

In the respiratory tract allergic rhinitis and sinusitis, hyperplastic rhinitis, nasal polyposis (the complete ætiology is not known in this connexion—nasal polypi may be entirely caused by bacterial infection or be the result of a sensitivity to the protein in these bacteria) and bronchial asthma may occur. As is well known, a great many parts of the body may be involved in allergic reactions, and the skin is possibly the commonest after the respiratory tract, or perhaps is even more commonly involved.

Ætiology and Pathology of Allergic Rhinitis and Sinusitis.

Some of the tissue changes caused by allergy are temporary, functional and reversible; others are organic and irreversible.

In the so-called immediate skin reaction in skin testing, in which a weal is obtained within a few minutes of the injection of an extract containing an antigen, the main tissue changes are œdema and hyperæmia; these are a reversible and temporary reaction. This is the type of reaction which occurs in urticaria, angioœdema and hay fever (allergic rhinitis) and in some forms of asthma.

Nevertheless, if the œdema is of long standing, some secondary tissue hyperplasia may occur.

Antigens and allergens are usually a protein complex, but a carbohydrate molecule may be implicated. The protein can be a body protein, and it is combination with a body protein which takes place in sensitivity to such substances as penicillin, aspirin and such a constitutionally simple substance as formalin. In many cases of bacterial sensitivity the protein is associated with a polysaccharide. In fact, complex molecules in which polysaccharides are conspicuous either alone or combined with protein constitute the majority of allergens (Burnet, 1950). In tuberculosis it is a sensitivity to the specific tuberculo-protein.

Antigens or allergens must be absorbed to produce clinical effects, and they may be inhaled, ingested, injected, absorbed from external contact with the skin, or absorbed from areas of infection. The absorption of bacteria or their breakdown products, either from an area of acute infection or from a chronic focus, may produce any type of allergic disease, but especially allergic rhinitis and sinusitis, though in the two last-mentioned the focus of infection is usually in the respiratory tract. It is the protein component of the bacterium which is the basis of the allergic reaction. This type of absorption may be the least important, but it is also the type in which we know least about the mechanism both of absorption and of reaction.

In an allergic subject the whole of the mesenchymal tissue is hypersensitive, but to a varying degree for each tissue, and the tissue showing the greatest sensitivity is referred to as the shock tissue.

The symptoms referable to this tissue are those to which attention is drawn and which suggest the name for the particular syndrome. Hence if the shock tissue happens to be in the mucous membranes of the nose and sinuses the disease is known as allergic rhinitis and sinusitis, and if it is in the bronchial mucous membrane, bronchial asthma.

The pollens causing hay fever are mainly air-borne; the insect-borne pollens do not usually come in close contact with humans.

Because the inhaled antigen reaches the surfaces of the nasal and bronchial mucosa first and in greatest concentration, the mucosa in these areas becomes sensitized first and it then tends to be the area where there is the greatest concentration of specific antibodies.

In perennial allergic rhinitis and sinusitis the antigen may be inhaled in dusts or fumes or gases, or be absorbed by ingestion or from an infective focus. The mechanism in the case of inhaled antigen is the same as that described under hay fever, but in the other two instances the shock tissue for some as yet unknown reason sometimes is in the nasal and nasal accessory sinus mucosa.

The histo-pathological changes in this mucosa in each case are similar, though of course with some antigens only seasonal in incidence. There is an increased capillary permeability, which is followed by œdema of the mucous membranes of the nose and accessory sinuses, to a lesser extent of the mucosa of the naso-pharynx, and also in some cases of the conjunctival sac. This œdema of the mucosa accounts for its bluish-grey, boggy appearance; this is the "wet" allergic type of mucosa recognizable clinically. Sneezing and lachrymation are present, and in some subjects swelling of the conjunctiva. Moreover, in some cases this pathological condition is transitory and reversible, appearing and disappearing as contact with the specific allergen is intermittently made. When this contact is continuous the œdema in time becomes permanent and fixed tissue hyperplasia may occur.

Cooke thinks that in those cases in which the allergy is the result of bacterial sensitivity the bogginess of the mucosa of the nose and sinuses may become permanent and extensive, and that it is in these subjects that the formation of polyposis occurs. From clinical experience I find myself in agreement on this point. However, Cooke distinguishes between this type of mixed infective and allergic condition, in which the infection came first and the allergy is secondary, and the type in which the allergy

was first (usually from sensitization to air-borne inhalants and to foods), and because of the local swelling and œdema with consequent inadequate ventilation and drainage a secondary bacterial invasion has occurred, causing a superimposed inflammatory state. The blue-grey, boggy mucosa takes on a reddened, engorged appearance, the serous discharge may become purulent, and where eosinophile cells predominated, now polymorphonuclear cells are in the majority.

In the former type, in which the allergy is secondary to sensitization to bacteria and the infection is of a minor relatively avirulent form, it is common for patients to state that they seldom suffer from the acute and recurrent upper respiratory infections with purulent and muco-purulent secretion described as "winter colds" by the laity. In my experience this group of patients develop nasal polypi. There is also another mixed group of cases in which sensitization to the non-infective allergens and to bacterial allergens coexists.

Shambaugh (1939) found in 65% of his asthma patients with an associated nasal allergy, thickening of the Malpighian layer of the epithelium with some eosinophilic infiltration. This, however, is only evidence of an irritated epithelium and is often found in chronic inflammatory infections. Nevertheless, combined with the œdema of the interstitial space in allergic states there is often gross infiltration with eosinophile cells, and predominant eosinophilia in the polymorphonuclear infiltration does point to an allergic element. Notwithstanding this, a high percentage of eosinophile cells in the nasal secretion does not always indicate allergy, and many allergic patients do not show pronounced eosinophilia. Hence nasal smear examinations are of little value.

This infiltration with eosinophile cells is found mainly in the stratum between the basement membrane and the glandular layer, and most of the œdema occurs in this area also. However, there still exists considerable difference of opinion in regard to the ætiology of the hyperplastic and polypoid changes found in a large percentage of patients suffering from allergy.

Grove, quoted by Cooke (1947), states that in his experience almost 100% of the pathological manifestations of allergic rhinitis and sinusitis are of the hyperplastic or thickened membrane type, differing from the suppurative type in the histological picture. There may be exacerbations of infection, when pus appears in the washings and neutrophile cells predominate in the submucosa, but these are only temporary. The true hyperplastic membrane is greatly thickened, often infolded, and tending to papillary formations and also polypoid formation.

There are areas in which the cilia are lost and varying degrees of metaplasia of the epithelium have occurred, though these two are not prominent features in the pure allergic type. The basement membrane is thickened, and often there is some hyalinization of this layer; in the *tunica propria* there are the gross œdema mentioned before, an increase in the fibrous tissue elements, and a small round-cell, plasma cell and eosinophile polymorphonuclear leucocyte infiltration. Neutrophile polymorphonuclear cells appear in infective exacerbations, but are rare otherwise. Grove found 30% to 35% of his allergic rhinitis patients to be suffering from polyposis, and Kelley (1936) in 100 cases of bronchial asthma found polypi in 26%.

Grove and Cooke (1933) examined by culture the lining membranes in this particular type of hyperplastic sinusitis removed at operation and found one or more organisms in 80% of cultures. Only one of 29 specimens of antral membrane was sterile, and 16 of 51 ethmoidal and sphenoidal specimens were sterile. They explained the latter larger percentage as being due to the fact that the specimens examined were mucous and cystic polypi, the contents of which are usually sterile, the infection being in the cyst wall or underlying membranes. Thirty-four of the positive cultures yielded a single organism; this, if confirmed in other series, is important. They found that of 87 specimens of membrane cultured the organisms grown differed in 43·7% from any recovered pre-operatively from diagnostic lavage and culture. Grove and Farrior (1940), in a bacteriological study of cultures of 108 specimens of antral membrane, found a staphylococcus in 57%, *Streptococcus viridans* in 43%, a pneumococcus in 39%, a hæmolytic streptococcus in 25%, an indifferent streptococcus in 3·7%, *Neisseria catarrhalis* in 3·7%, *Proteus vulgaris* in 0·9%, a diphtheroid bacillus in 0·9%, and *Escherichia coli* in 0·9%; 4% of specimens gave negative results. However, Kistner (1931) found streptococci in 94·5% of 400 sinus membrane specimens examined by culture.

Hansel (1929) and others believe that organisms in these membranes are secondary invaders; but Kistner (1931), Goodale (1922), Grove (1941) Grove and Cooke (1933), and Grove and Farrior (1940) do not agree.

This is possibly the most important point in the consideration of this problem. Are these cases of hyperplastic polypoidal rhinitis and sinusitis ever truly allergic alone? It is doubtful; the infective element may be the causal agent in the polyposis. The question is as yet unanswered, and the successful and correct treatment would appear to depend upon this answer.

Grove, after careful analysis of his own cases, thinks that there has been a good deal of confused thinking and writing about the results of surgical treatment. Surgical treatment in recent years has fallen into disfavour to some extent, especially because of indifferent results in relieving asthma. Grove holds that to estimate correctly the effect of surgical treatment of the sinuses on asthma two points should be considered: (*a*) whether there is infection alone or infection associated with an allergy of the skin-sensitive type, (*b*) whether the diseased membranes have been completely or incompletely removed by operation. He maintains that associated allergies must be treated properly, extranasal infections attended to and complete eradication of all sinus infection carried out, or good results cannot be expected. Follow-up studies of his own cases have shown that with increasing post-operative periods of time steadily improving results are obtained. He believes this to be due to the gradual overcoming of secondary infections in the cervical and bronchial lymph nodes and bronchial mucosa present at the time when the sinus disease is dealt with. The elimination of this infection gradually follows. Autogenous vaccines are considered by Grove to be helpful in the treatment of infective asthma.

Vasomotor Rhinitis.

There certainly has been a good deal of confused thinking and writing in the literature in connexion with vasomotor rhinitis.

Some authors apparently regard perennial allergic rhinitis and vasomotor rhinitis as synonymous. If such an interpretation is accepted, the discussion in the preceding section is adequate. I believe that if such was the case the term would be better dropped. However, in clinical experience there is a group of cases which present many, if not all, of the symptoms and signs of allergic rhinitis, but in which no allergen can be detected by skin tests, scratch or intradermal, or by elimination food tests, nor can the sufferer associate any particular offending substance which may contain an allergen with the onset of symptoms.

The obvious explanation in many of these cases is that in our incomplete state of knowledge of the subject, with consequent inadequate means of detection of allergens, an undiscovered one or more of these substances are responsible. It must be remembered that the skin is only one "shock organ" and the manifestations of the antigen-antibody reaction may occur in some organ or organs deeper within the body with not so obvious symptoms and a corresponding greater difficulty in applying diagnostic tests such as are used on the skin.

Another group of patients persist in their opinion that sudden change of temperature in their environment brings on the syndrome of watery nasal discharge and sneezing, others that certain irritating gases or fumes do, the condition being described as a chemical irritation; but the question of an allergen has to be excluded in these cases also—for example, in the case of formalin sensitivity.

XUM

An endocrine factor has been postulated—that the allergic nasal syndrome may be precipitated in patients who have hormonal deficiencies, or at least who have some degree of endocrine imbalance. There is no definite proof of this hypothesis, but some case histories suggest that it is possibly true. Occasionally a woman develops an allergic type of rhinitis during pregnancy for which no allergen may be found and which disappears after the baby is born; or the condition may appear during the menopause and after a variable period of time disappear as suddenly as it developed. Our knowledge of the anatomical pathways of the reflex arcs which may be concerned in the production of nasal vasomotor phenomena caused by the different factors just described is incomplete to say the least.

These reflex arcs are composed of afferent and efferent components. The set-up in the case of the fifth nerve, which provides most of the afferent components, is complicated by the fact that many sympathetic afferent fibres use the branches of the trigeminal nerve as pathways for varying parts of their course, and stimulation of the nerve may produce effects via the sympathetic efferent component of the arc. Possible effects are dilatation of the blood vessels of the nasal mucosa, including the erectile tissue associated with the turbinates, and an increase in the glandular secretions of the mucosa; sneezing, nasal obstruction and a watery discharge are consequent symptoms.

It is obviously difficult to determine which of the many possible anatomical pathways is used by afferent impulses—whether they reach the central nervous system via the sympathetic sensory nerves or via the somatic (fifth) sensory nerves.

In a small group psychic states seem to produce the allergic type of syndrome, and there is a possibility that there is a connecting efferent component between the cortex and the nasal mucosa via the sympathetic efferent fibres causing vasodilator phenomena, comparable to that which occurs in the external genitalia under psychic stimulus.

The determination of the pathway of the afferent component of the reflex arc in the endocrine imbalance group is even more difficult. One may postulate that vasomotor rhinitis is a condition caused by an upset in the normal balance of the nerve supply to the smaller blood vessels in the nasal and sinus mucosa.

SUMMARY.

1. The surgical pathology of the closely allied non-specific rhinitis and sinusitis is dealt with more or less together.

2. Non-specific rhinitis and sinusitis can be classified as follows: (i) acute rhinitis and sinusitis; (ii) chronic rhinitis and sinusitis: (*a*) simple chronic, (*b*) chronic hypertrophic (synonym, chronic hyperplastic), (*c*) chronic purulent—polypoid, sclerosing or fibrotic, glandular or adenomatous, papillary, atrophic; (iii) allergic rhinitis and sinusitis, and vasomotor rhinitis.

3. Acute rhinitis and sinusitis, or inflammation of the mucosa lining the nose and accessory nasal sinuses, occurs at the onset of a number of diseases, including the common cold, influenza and some of the exanthemata, especially measles. The initial stage is probably the result of invasion of the tissues of the area by a virus, which causes enough interference with the normal defensive mechanism to allow the entry of secondary pyogenic invaders. These latter organisms cause the main subjective symptoms. Predisposing causes are listed. The histo-pathology is then described in detail, and finally the pathology in relation to symptoms, complications and treatment is discussed.

4. The classification of chronic rhinitis and sinusitis endeavours to group a set of conditions which are obviously interrelated. (i) Simple chronic rhinitis is a catarrhal inflammation; the secretion is serous, not purulent. Swelling and bogginess of the epithelium and œdema and hyperplasia of the subepithelial tissues are present, as well as an increase in the amount and character of the secretion. It occurs (*a*) as a result of incomplete resolution of acute rhinitis; usually there is a predisposing factor; (*b*) in chronic low-grade sinus infection; (*c*) in children, associated with nasal obstruction due to enlarged adenoids; (*d*) in certain occupations involving irritation of the nasal mucosa. Because of continued or repeated congestion of the vascular apparatus the vessels become permanently dilated to a varying degree. There are also increased permeability of these vessels, an increase in the fibroblastic content of the submucosa, and hyperplasia of the epithelium, including an increase in the goblet cells; the glandular content of the submucosa is also increased. All this leads to nasal obstruction and to an excess of seromucinous nasal discharge. The pathology in relation to symptoms, diagnosis and treatment is discussed. (ii) Chronic hypertrophic rhinitis is the end stage of simple chronic rhinitis, in which there occur greater fibrosis and almost complete loss of contractility of the vessel walls, which are greatly thickened. Often papillary hypertrophy of the ends and lower borders of the turbinates is present, because of the further increase in hyperplasia of the epithelium. Nasal obstruction is greater, sense of smell is interfered with or often lost. The nasal secretion is often thicker, stickier and more tenacious; saprophytic bacterial growth may occur because the secretion is retained for too long and gives an unpleasant odour. Whereas in simple chronic rhinitis vasoconstrictors still can act and shrink up the mucosa, and a probe will indent the boggy tissue and the mark will disappear fairly quickly, in this condition vasoconstrictors have little effect and the indent of a probe is less and slow to disappear. (iii) Chronic purulent rhinitis and sinusitis begin as acute rhinitis; complete resolution does not take place, usually in one or more of the accessory nasal sinuses, and subacute and finally chronic inflammation supervenes. As the condition persists and progresses, certain definite histopathological changes take place in the mucosa lining the particular sinus affected. The reaction of the mucosa and submucosa to chronic inflammation as a result of bacterial infection varies. Why it varies in different subjects and in different circumstances has not been fully worked out. The matter is briefly discussed. The various types of histo-pathological reaction in the tissues are used as a basis for classification. The name of the most obvious feature is given to the different groups—for example, polypoid, fibrotic, glandular, papillary and atrophic types of chronic purulent rhinitis and sinusitis. The details of these different groups are described and discussed.

5. Allergic rhinitis and sinusitis and vasomotor rhinitis are discussed. A brief outline of the development of the principles and theory of allergy is given.

6. The ætiology and pathology of allergic rhinitis and sinusitis are described. The vasomotor rhinitis group and syndrome are discussed.

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THE CAUSE OF HYPOKALÆMIA (HYPOPOTASSÆMIA) FOLLOWING PARA-AMINOSALICYLATE THERAPY.

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HYPOKALÆMIA has been found as a complication of PAS (para-aminosalicylate) therapy by Cayley (1950) and by Heard *et alii* (1950). Also Summers (1949) and Hall (1950) have observed patients receiving PAS in whom the clinical findings provide presumptive evidence of hypokalæmia. We have observed additional cases during an investigation of the problem.

As PAS has a definite place in the treatment of tuberculosis it is extremely important that its use should be free of such a dangerous complication as hypokalæmia. An investigation has been carried out to determine the cause of hypokalæmia and to indicate means by which its occurrence may be prevented.

Possible Causes of Hypokalæmia.

In an earlier investigation at this hospital (Heard *et alii*, 1950) hypokalæmia had been produced by an aqueous solution of sodium para-aminosalicylate, flavoured with liquorice. It had been noted that the PAS was coloured by a small amount of dark impurity. Isolation of all the impurities present has not been possible, but investigations have shown that it is possible to extract a dark-coloured impurity and to measure the amount present in any sample of the drug. One commercial preparation of PAS which had caused hypokalæmia previously was found to contain up to 1% of this impurity. It was suspected that this, or other impurities associated with it, could have been the cause of hypokalæmia.

Extractum Glycyrrhizæ Liquidum (British Pharmacopœia) had been used as a flavouring agent, four drachms being administered daily. It was found that this contained about 40% of solids. The amount of dried liquorice extract given daily would be about 5·7 grammes.

Using 20 to 45 grammes of *Succus Liquiritiæ* daily, Molhuysen *et alii* (1950) found that this lowered the serum potassium level appreciably. Although a smaller dose of liquorice was used to flavour PAS, it was evident that the effect of liquorice would have to be observed.

When sodium para-aminosalicylate is administered, two grammes of sodium are ingested with every twelve grammes of PAS. It was possible that the daily ingestion of this quantity of sodium might lead to an increased excretion of potassium, thereby producing hypokalæmia. It was also possible that PAS itself might cause the complication.

Methods of Investigation.

The subjects used were all patients with tuberculosis for whom chemotherapy was deemed advisable. All patients were given 12 grammes of PAS daily for six days per week. The usual course was of 12 weeks' duration; in some cases it was of shorter duration. The PAS was administered as the potassium salt in aqueous solution.

The first series of patients was given the commercial preparation containing impurities. This has been designated impure PAS. Some of these patients received liquorice (four drachms daily) as a flavouring agent; others received the PAS flavoured with syrup. According to medical indications a proportion of the series received streptomycin in addition to the PAS.

The second series was similar, except that another preparation of PAS was used which was much purer and contained only 0·05% of the dark impurity.

To study effectively thei nfluence of the impurity and liquorice and to eliminate any influence exerted by streptomycin it has been necessary to divide the patients into eight groups.

The mean of duplicate serum potassium readings (estimated to the nearest half-milligramme per 100 millilitres) was calculated from samples of blood taken at regular intervals from all patients during each PAS course. For each group of patients studied these estimations were divided into those which were 16 milligrammes *per centum* or more and those below 16 milligrammes *per centum*. The proportion of serum potassium estimations below 16 milligrammes *per centum* was then used as a means of assessing the influence of impure PAS, liquorice and streptomycin.

The figure 16 milligrammes *per centum* was arrived at by estimating the mean and standard deviation of 76 serum potassium estimations from 73 tuberculous patients not receiving PAS and then by selecting as the lower level of the normal range a figure which in whole numbers was three times the standard deviation below the mean. (The mean = 17·6 milligrammes, the standard deviation = 0·4 milligramme; the mean − (3 × standard deviation) = 16 milligrammes in whole numbers.)

An additional assessment of the results was made by examining the number of patients in the different groups whose serum potassium levels dropped below (*a*) 16 milligrammes *per centum* and (*b*) 14 milligrammes *per centum*.

Results.

The Effect of Impurity.

The results obtained when impure PAS was used without liquorice have been compared with those produced by the purer PAS without liquorice.

The serum potassium level was frequently lowered when impure PAS was used; the incidence was considerably less with pure PAS. Independently of the use of streptomycin there is a highly significant difference between the two groups (see Table I).

The Effect of Liquorice.

When purer PAS was used the serum potassium level was lowered more frequently in the groups receiving liquorice. This increased incidence was statistically significant (see Table II).

It was not possible to show that liquorice enhanced the effect of the impurity. The incidence of lowered serum potassium levels was not significantly different in the group receiving impure PAS with liquorice and the group receiving the same preparation without liquorice (see Table III).

Similarly when liquorice was used there was an insignificant difference in the incidence of lowered serum potassium levels when the effects of impure and pure PAS were compared (see Table IV).

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Streptomycin.

No definite influence attributable to streptomycin was found.

Sodium.

The finding that hypokalæmia could develop when potassium PAS was administered has shown that the ingestion of sodium was not an important factor.

TABLE I.

Effect of Impure PAS (without Liquorice).

Material Administered.	Serum Potassium Estimations.		
	Number below 16 Milligrammes *per Centum.*	Number 16 Milligrammes *per Centum* or More.	Total.
Impure PAS with streptomycin	8	9	17
Purer PAS with streptomycin	18	122	140
Impure PAS without streptomycin	8	12	20
Purer PAS without streptomycin	0	18	18

With or without streptomycin, there is a relatively greater number of low potassium estimations with impure PAS in comparison with purer PAS.

$\chi^2 = 10{\cdot}5$ (with streptomycin), $\chi^2 = 6{\cdot}9$ (without streptymycin); this is significant in both instances.

Assessment of the Results on a Case Basis.

It was of interest to ascertain whether the impure PAS caused the serum potassium level to fall in a greater proportion of patients than did purer PAS.

A comparison was made between the group of patients receiving impure PAS without liquorice and the group receiving purer PAS without liquorice. A proportion of the patients in each group received streptomycin, but as no definite influence attributable to streptomycin had been detected it was convenient to ignore it in this comparison.

TABLE II.

Effect of Liquorice (with Purer PAS).

Material Administered.	Serum Potassium Estimations.		
	Number Below 16 Milligrammes *per Centum.*	Number 16 Milligrammes *per Centum* or More.	Total.
Purer PAS with streptomycin *plus* liquorice	11	30	41
Purer PAS with streptomycin and without liquorice	18	122	140
Purer PAS without streptomycin *plus* liquorice	7	9	16
Purer PAS without streptomycin and without liquorice	0	18	18

With or without streptomycin there is a relatively greater incidence of low potassium estimations with liquorice compared with the group without liquorice.

With streptomycin $\chi^2 = 4{\cdot}60$, without streptomycin $\chi^2 = 7{\cdot}4$; this is significant in both instances.

In conformity with the previous results, it was found that, compared with the patients receiving purer PAS, a relatively greater number of those receiving impure PAS developed a serum potassium level below 16 milligrammes *per centum*; similarly the group receiving impure PAS had a relatively greater number of cases with a serum potassium level of 14 milligrammes *per centum* or less. As is seen in Table V, the difference in the incidence of hypokalæmia is significant. The data were examined to ascertain the effect of liquorice on individual patients. It was found that, of the patients receiving purer PAS, those receiving liquorice were more likely to develop a serum potassium level below 16 milligrammes *per centum* than were those not receiving liquorice.

TABLE III.

Test of Reinforcement of Impurity Effect by Liquorice.

Material Administered.	Serum Potassium Estimations.		
	Number Below 16 Milligrammes *per Centum.*	Number 16 Milligrammes *per Centum* or More.	Total.
Impure PAS with streptomycin and liquorice	21	48	69
Impure PAS with streptomycin and without liquorice	8	9	17
Impure PAS without streptomycin and with liquorice	17	14	31
Impure PAS without streptomycin and without liquorice	·8	12	20

In both cases the difference is not significant.

The difference between the groups is not quite significant (see Table VI). This can be attributed to an insufficient number of cases, as the previous more sensitive method of assessing the results has revealed that liquorice caused a depression of the serum potassium level.

There was no significant difference between the group with and the group without liquorice in the number of patients developing a serum potassium level of 14 milligrammes *per centum* or less.

TABLE IV.

Test of Reinforcement of Liquorice Effect by Impurity.

Material Administered.	Serum Potassium Estimations.		
	Number Below 16 Milligrammes *per Centum.*	Number 16 Milligrammes *per Centum* or More.	Total.
Impure PAS with streptomycin and with liquorice	21	48	69
Purer PAS with streptomycin and with liquorice	11	30	41
Impure PAS without streptomycin and with liquorice	17	14	31
Purer PAS without streptomycin and with liquorice	7	9	16

The differences are not significant.

Discussion.

The results indicate that both an impurity in the PAS and liquorice will lower the serum potassium level; the evidence does not demonstrate any reinforcement of the impurity effect by added liquorice. However, the lowest serum potassium levels were encountered when impure PAS was administered with liquorice, a finding suggesting that a combined effect of the two factors may have been operative. The drop in the serum potassium level caused by liquorice was slight and was seldom below 14 milligrammes *per centum*, whereas that caused by the impurity was greater and was frequently below 14 milligrammes *per centum*. This accounts for the significant finding for

the impurity effect and the absence of a significant finding for liquorice when the patients were divided into those that developed a serum potassium level of 14 milligrammes *per centum* or less and those that remained above this level. It was often necessary to prevent the further lowering of the serum potassium level, due to the impurity, by administering a potassium salt and suspending administration of PAS. This was necessary only once for the effect due to liquorice when used with purer PAS. We have noted evidence of a slight fall in the serum potassium

TABLE V.
Effect of Impurity on Individual Cases.

Material Administered.	Serum Potassium Estimations.		
	Number with Serum Potassium Level Falling to 14 Milligrammes *per Centum* or Less.	Number with Serum Potassium Level Remaining Above 14 Milligrammes *per Centum*.	Total Number.
Impure PAS without liquorice	5	5	10
Purer PAS without liquorice	2	27	29

The number of patients developing a low serum potassium level was relatively greater among those receiving impure PAS.
$\chi^2 = 6{\cdot}7$ (significant).

level with the purer PAS preparation without liquorice. This has been attributed to the small amount of impurity present in the purer material. These slight alterations in the level of the serum potassium have been found to be replaced by normal values spontaneously without suspension of the drug.

Further details concerning the nature of and tests for the impurity are published separately (Neufeld, 1951).

From the clinical aspect it was possible to show that when impure para-aminosalicylic acid was administered as

TABLE VI.
Effect of Liquorice on Individual Cases.

Material Administered.	Serum Potassium Estimations.		
	Number with Serum Potassium Level Falling to 14 Milligrammes *per Centum* or Less.	Number with Serum Potassium Level Remaining Above 14 Milligrammes *per Centum*.	Total Number.
Impure PAS with liquorice	9	4	13
Impure PAS without liquorice	10	19	29

The number of patients developing a low serum potassium level was relatively greater among those receiving liquorice.
$\chi^2 = 3{\cdot}1$. $P = <0{\cdot}10>0{\cdot}05$.

coated granules a fall in the serum potassium level occurred. This helped to eliminate the possibility that neutralization of the acid by alkalis produced some degradation product responsible for hypokalæmia.

It is clear that to prevent hypokalæmia as a complication only pure PAS should be administered. It is also inadvisable to use liquorice as a flavouring agent.

It is anticipated that when such precautions are taken it will be unnecessary to control the administration of PAS by regular serum potassium estimations. The use of the potassium salt of PAS is probably unnecessary, and from the viewpoint of hypokalæmia, provided the preparation is sufficiently pure, it would appear to be immaterial whether the sodium, calcium or potassium salt or the acid is used. We have given the calcium salt of PAS to eight patients without evidence of hypokalæmia. We are also using a reasonably pure sodium salt, which is proving satisfactory.

Summary.

1. It has been shown that hypokalæmia which has developed following PAS therapy is due to an impurity in certain commercial preparations of PAS.

2. This complication can be prevented by the use of sufficiently pure PAS.

3. To prevent avoidable deaths it is necessary for the commercial preparation of PAS to be subject to rigid standards of purity.

4. Liquorice causes a slight lowering of the serum potassium level and is to be avoided as a flavouring agent for PAS.

Acknowledgements.

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THE DETERMINATION OF IMPURITIES IN COMMERCIAL PARA-AMINOSALICYLIC ACID.

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OFTEN when a new drug is used, a number of complications occur, which are found on close investigation to be due not to the drug itself, but to impurities contaminating the drug.

Naturally, a certain time period elapses before a definite purity standard can be set up for a new drug and before such a standard can be made official.

If the drug is comparatively simple to prepare, its commercial exploitation will attract many less experienced drug manufacturers, and in consequence material may come on the market which is dangerous to the patient.

Clinical experience in this hospital and reports from overseas have clearly indicated that in some cases PAS may cause potassium deficiency in the blood serum, leading to death if careful precautions are not taken.

A large range of commercial samples of PAS was therefore compared, and a number of qualitative and quantitative tests for certain impurities are suggested, which will indicate material of poor quality.

The methods so far suggested in the literature are not suitable to determine small amounts of impurities in PAS. They aim at the determination of the amount of PAS itself in a sample, the amount of impurities being arrived at by subtraction. Very small errors in the determination of the amount of PAS, however, will be magnified out of all proportions if the absolute amount of impurity is rather small.

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As, however, the doses of PAS are so large (12 grammes per day), even relatively small amounts of impurities may have very severe effects.

The following methods are therefore based on a direct determination of the impurities. Like all the usual routine methods which can be carried out rather quickly by relatively unskilled operatives with simple equipment, it does not claim to cover the complete range of all the endless number of possible impurities.

The methods suggested aim at the determination of certain impurities which were found to contaminate the commercial products under investigation. In particular they determine with accuracy a dark-coloured, ether-insoluble compound, which was found to be present in those PAS preparations which were associated with potassium deficiency. It was largely absent from the preparations associated with symptom-free administration.

While sufficient work has not been done yet to ascertain whether this impurity is the direct cause of the hypokalæmia, the indications are that freedom from this impurity in PAS seems important.

The exact chemical nature of this coloured compound has not been investigated, but it appears to be an oxidation product, possibly in a polymerized form of meta-aminophenol. This is easily formed when para-aminosalicylic acid is exposed to heat or the influence of acids.

Other impurities determined by the suggested method are phenols and easily carbonizable impurities.

There is very little danger that a product complying with the following suggested tests could be dangerous in regard to hypokalæmia. The tests are simple enough to be carried out in any pathological laboratory.

Experimental Methods.

Qualitative Test for Readily Carbonizable Substances.

A qualitative test for relatively carbonizable material is carried out by dissolving 10 milligrammes of the PAS sample in two millilitres of cold concentrated sulphuric acid. No more than a light yellow colour is produced, and there should be no bluish fluorescence noticeable in reflected light.

Colorimetric Comparison of PAS Samples.

To make a colorimetric comparison of PAS samples a solution is prepared containing 3% PAS. The colour intensity is compared with the colour of a 5% ferric ammonium sulphate solution.

If free acid is to be tested, the weighed amount is moistened with a little water, and enough 10% sodium hydroxide solution is added carefully from a pipette until all has just dissolved. A flattened glass rod is used to break up any small lumps of substance. The solution is then made up to volume. A water-soluble salt is just dissolved in the correct amount of water.

The colorimetric comparison should be carried out immediately, as the PAS solutions quickly darken in dilute concentrations. The PAS solution should be lighter in colour than the empirical standard. A good sodium PAS solution should not be darker than a 1% ferric ammonium sulphate solution. A green filter may be used, and a photoelectric colorimeter is preferable to a visual one.

Estimation of Ether-Insoluble Dark Impurity.

To determine the amount of the ether-insoluble dark impurity present, one grain of the acid is dissolved in 50 millilitres of ether with the aid of a flattened glass rod to break up lumps of material. If there is a dark brown, flocculent precipitate, this is spun down in portions in a conical centrifuge tube of 10 to 15 millilitres capacity.

The precipitate is washed with several small portions of ether, with the aid of a small glass rod (one to two millimetres thick), and the ether is decanted after spinning. The precipitate is weighed after drying at incubator temperature and should not amount to more than 0·05% of the PAS (five milligrammes).

If a salt of PAS is to be tested, two grammes of the salt are dissolved in five to ten millilitres of water. The solution is placed in a separating funnel, and 100 millilitres of ether are added.

A 10% acetic acid solution is added in portions, each addition being followed by short shaking, to take up the precipitated acid into the ether. Addition is stopped when the pH of the aqueous solution is about 4·0.

Any dark precipitate collecting at the interphase between the aqueous and ether layer is collected and weighed.

Estimation of Meta-aminophenol.

The method of estimating meta-aminophenol is based on the fact that PAS as an acid forms an ether-insoluble salt with bicarbonate, whereas meta-aminophenol at this pH (7·5-7·8) remains the free phenol and can be extracted into an organic solvent like ether.

Ten grammes of PAS (free acid) are dissolved in a slight excess of saturated sodium bicarbonate solution. If a water-soluble salt is to be tested, this is dissolved in water and a small amount of sodium bicarbonate is added.

The aqueous solution is shaken three times with ether. The bulked ether solutions are shaken back with two small amounts of sodium bicarbonate solution.

The ether solution is dried with a little anhydrous sodium or magnesium sulphate, and the ether is removed by distillation at a low temperature.

The residue is weighed after being dried in an incubator for a few minutes.

The amount of meta-aminophenol should be negligible, certainly less than 10 milligrammes from 10 grammes of PAS.

Melting Point.

Para-aminosalicylic acid melts undergoing decomposition, and the melting point varies too strongly with rate of heating and small amounts of moisture content to be significant for the degree of purity.

Immediately after melting a good sample of PAS appears in the melting-point tube as a colourless or pale straw-coloured liquid; poor samples go dark brown and often are not clear.

The melting points of salts of PAS are not at all sharp, and discoloration commences far below the melting point, even in pure samples. They are of no importance as a means of checking purities.

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Reports of Cases.

BLACKWATER FEVER APPARENTLY PRECIPITATED BY PROGUANIL.

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THE patient concerned is a white man, aged twenty-seven years, a carpenter. He normally enjoys good health, and there is nothing relevant in his past history except a few attacks of malarial fever, the last more than six months ago. At 1.30 p.m. on February 1, 1951, he suffered a severe rigor, was thereafter "very hot" and at 5 p.m. commenced spontaneously to sweat. From soon after the onset of the rigor he had a sharp ache over the sacrum and lower lumbar region; late in the afternoon he passed some dark urine.

I was called to see him at 8 p.m. on account of the backache, which was causing him very great discomfort. His temperature and pulse rate were normal; his abdomen felt normal, with no tenderness over liver or spleen; by artificial light his conjunctivæ did not seem jaundiced (and were normal by daylight next morning); there was no tenderness in the costo-vertebral angles. The only abnormal signs found were the presence in scanty numbers of *Plasmodium falciparum* in his blood and a dark smoky-brown urine with much amorphous debris, reacting strongly to

the guaiac test. His general condition was excellent. The diagnosis was obviously blackwater fever, probably of the mild intermittent type.

Inquiry revealed that while nominally supposed to be taking 0·1 gramme of proguanil hydrochloride (as "Paludrine") daily, he was actually very irregular in taking it. His home is in Wau, which is only moderately malarious, but about eighteen days previously he had spent some days in Lae, which is a hyperendemic area. In Lae he had slept in an unscreened house without a net, and had taken no "Paludrine". He had taken 0·1 gramme of "Paludrine" perhaps four or five times in the week preceding this present attack, and when the rigor started he took 0·2 gramme. As for quinine, he stated that some two months previously he had temporarily run out of "Paludrine" and had taken five grains of quinine sulphate daily for two weeks. He was quite definite that he had taken no quinine for six weeks prior to this attack—in fact, he stated categorically that he had had no medicines of any kind whatever, even aperients or aspirin, for months, except the quinine and "Paludrine" as outlined above.

In accordance with the principle that the apparent precipitating drug must be avoided in treating blackwater fever, he was given 0·6 gramme of amodioquin hydrochloride (as "Camoquin") daily for four days; examination of thick blood films each morning for five days showed no parasites. He was also given an alkaline mixture and increased fluids; his urine production was excellent. Successive samples checked during the second day showed progressively less blood and debris, and on the morning of the third day there was neither blood nor debris, but still a heavy cloud of albumin. By the seventh day the albumin had also disappeared. His backache was much easier on the second day and had quite gone by the fourth day. On the seventh day he was feeling perfectly well, and insisted on returning to work. He has been taking chloroquine diphosphate (as "Aralen") prophylactically since, and has had no sign of any recurrence, although in insufficiently treated cases there is invariably a recurrence within seven to ten days.

Discussion.

The mild form of blackwater fever with intermittent hæmoglobinuria is known to occur in New Guinea (Gunther, 1936). In this form, with the emergence of each swarm of merozoites there is a single restricted occurrence of hæmolysis, sufficient to produce obvious hæmoglobinuria, which clears more or less completely by the time of the next rigor. This goes on until all circulating parasites are destroyed, either by treatment or by becoming involved in some way in the processes of hæmolysis. In the present case it would seem that since the "Camoquin" promptly destroyed all circulating parasites (as suggested by the blood films, examination of which showed that the parasites survived the original hæmolysis but disappeared after "Camoquin" was given), the disease was aborted.

This case is reported primarily to put on record the onset of one form of blackwater fever when the only drug taken was proguanil hydrochloride, which if any drug is to be regarded as precipitating the disease, must have been the precipitating agent. But the drug had been taken irregularly and in quite the wrong way, so there is no indication here of its value as a prophylactic. This case may also be regarded as illustrating the prompt action of "Camoquin" (parasites persist usually for four, sometimes for five days with proguanil and "Atebrin"); it is not suggested that "Camoquin" is a specific for blackwater fever, but it is emphasized that a rapidly acting drug can cut short this particular type of blackwater fever by its effect in removing the parasites—quinine would probably have been equally effective in this case, and equally safe, since it was not the precipitating agent.

Reference.

Gunther, C. E. M. (1936), "A Case of Blackwater Fever Showing Intermittent Hæmoglobinuria", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 542.

CONGENITAL ŒSOPHAGEAL ATRESIA WITH TRACHEO-ŒSOPHAGEAL FISTULA.

By ERIC GOULSTON,
Sydney.

(From the Royal Alexandra Hospital for Children.)

EXCESS MUCUS with choking and cyanosis after feeding attempts in the newborn means œsophageal atresia until proved otherwise. Awareness of this condition should lead to its earlier recognition, for the chances of success at operation are greatly enhanced by early suspicion of its presence.

Some developmental anomalies offer a fascinating challenge in their study as to ætiology and management. Such a condition is œsophageal atresia, incompatible with life unless relieved. Gray Turner regards its frequency as one in every 2000 births, while other authors place the incidence as high as one in 800 births. It is obvious, therefore, that many such cases in the past have been regarded as bronchopneumonia or intracranial birth injuries. Associated anomalies are fairly common, and in the case reported below a recto-vaginal fistula was present.

FIGURE I.

Embryologically there is a faulty development about the six to ten millimetre stage and it is considered that prenatal environmental and not genetic influences are causative factors. The diagnosis is made by finding obstruction to the passage of a small catheter down the œsophagus and confirmed after injecting one millilitre of lipiodol. The typical picture is shown by the accompanying X-ray film (Figure I), which portrays the common variety of a blind upper pouch, with spill-over into the lungs and gas in the stomach. It is obvious that barium should never be used as the opaque material, and it is wise to aspirate the lipiodol.

Clinical Record.

A female child, aged three days and weighing five pounds eight ounces at birth, was admitted to the Royal Alexandra Hospital for Children, Camperdown, on January 19, 1951, being referred by Dr. E. Lorger, of Parkes, New South

Wales. The presenting symptoms were inability to take feedings owing to regurgitation and coughing spasms with pooling of secretions in the pharynx. The mother's pregnancy and labour had been uneventful, and in the immediate neonatal period the child had appeared normal. The above-mentioned symptoms occurred from the time the first feed was attempted. There was no history of cyanotic attacks.

On examination the child's general condition was fair with a moderate degree of dehydration. A mild form of bronchopneumonia was present, the abdomen was flat and soft, and the baby had a vaginal anus. A catheter was unable to be passed into the stomach, and an X-ray examination after the injection of three-quarters of a millilitre of lipiodol revealed a blind upper pouch.

Intravenous routine pre-operative preparation was commenced, and operation was performed fifteen hours after the child's admission to hospital. At operation, with the patient under "closed" intratracheal ether anæsthesia, administered by Dr. C. Sara, a Vogt IIIb type of abnormality was found; an end-to-end anastomosis was obtained and the fistula closed. The intratracheal tube, inserted for administration of the anæsthetic, was left in place for suction purposes for twelve hours and then removed. The child was nursed in a steam tent and given oxygen for forty-eight hours and maintained by continuous intravenous therapy wholly for the next five days. On the fifth day a small feed of sterile glucose solution coloured with methylene blue was given. This was taken slowly without any untoward incidents. The continuous intravenous therapy was maintained till the eleventh post-operative day; during this time the child's oral feeding was established with pooled expressed breast milk, which was readily taken. The child has since thrived and gained weight regularly.

Discussion.

In few other conditions are the hazards of successful treatment greater or the margin for error less. After pre-operative preparation, the optimum time for operation must be carefully chosen. Hydration is not a serious factor in the early days of life, and it is better to keep the baby slightly dehydrated than to risk the ill-effects of over-hydration. Routine procedures include the adoption of a head-low posture with frequent turning from side to side, continuous oxygen administration, gentle suction through a nasal catheter, chemotherapy, blood examination, the correction of dehydration if necessary, and the exhibition of vitamin *K* in the presence of jaundice. One then decides whether the general condition can be further improved by waiting longer, but delay beyond twenty-four hours is seldom of avail as the continuance of aspiration pneumonia outweighs any further gain. Sedation is not required, and with the minimum of handling and with use of the usual methods of preventing heat loss, the baby is transferred to the operating theatre.

The advantages of light intratracheal anæsthesia are many, and the role of the anæsthetist is a major one in the team. The extrapleural right-sided approach by subperiosteal resection of the second rib is considered to give the best and most rapid exposure and to cause the least shock.

Post-operative care is also all-important and calls for special nursing attention. It includes attentive supervision of the intravenous drip therapy, steam-tent nursing, repeated pharyngeal aspiration, postural changing, chemotherapy and oxygen administration. There seems no need for a routine gastrostomy if a sound anastomosis has been obtained. The hazards of vomiting are by no means eliminated by gastrostomy feedings, and when excess mucus becomes less apparent, saliva is being swallowed. When there is no drainage from the drainage tube, it is safe to proceed cautiously with oral feedings.

Summary.

1. A lower mortality rate in œsophageal atresia can be assured by earlier diagnosis.

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The Medical Journal of Australia

SATURDAY, MAY 19, 1951.

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WALNUTS AND STEAM HAMMERS.

THERE are many ways of cracking walnuts, but no one in his right senses makes a habit of using a steam hammer for this purpose. Conceivably adjustments might be made in the steam hammer or safeguards introduced so that the shell of the nut would crack and the kernel would not be reduced to something flatter than the proverbial pancake. Such happenings would, however, be in the nature of an experiment or an exhibition and would not be every-day occurrences. The man who tried to wield the steam hammer every day might become proficient in its use, but every now and again he would do more harm to the unfortunate walnut than he intended. Further, he would expend a great deal of energy which might well be conserved and the cost of his undertakings in relation to his achievements would be unduly high. Dealing with different kinds of nuts, a man will use different methods to crack their shells. For the humble peanut he will use his fingers—he will not try to apply the dignified and reversible nut-crackers which grace his home equipment; when he comes to the coconut, he may cut it with a sharp knife or use some kind of blow on the coconut with something solid or with the coconut on something solid. In the practice of medicine there are many nuts to be cracked. They are found both in diagnosis and in treatment. Like those in the vegetable kingdom, they are of many different kinds. It is necessary to mention only two in order to show how variable in toughness medical nuts may be—the kidney-infection-diagnosis nut and the acute-and-chronic-infection-treatment nut. The point is that the medical investigator or therapist who would deal with the contents of medical nuts should find out how tough a nut is likely to be before he tries to deal with it.

Nowadays we have too much steam-hammer medicine, especially in treatment. Medicine as an art is being replaced by medicine in which understanding, judgement, subtlety and wisdom are pushed more and more into the background. This tendency must be deplored for several reasons. A practitioner who forsakes simplicity in order that he may "use modern advances to the full" is by his own hand restricting the field of his experience. He can learn little of the delicate susceptibilities of the body and of its finer reactions to some noxious agent. These have no time to show themselves if bludgeoning methods are preferred. The practitioner's erudition must suffer and when the time comes for him to rely on his own experience and judgement because for one reason or another he is deprived of his modern aids and cannot use his "steam hammer", though the condition of the patient really calls for the heavy method, the patient's welfare is affected. In this case the patient suffers because his medical attendant has no depth of knowledge. When the attendant is a steam-hammer doctor the patient may respond to heavy treatment that is not needed, only to find that in a subsequent illness when he really needs all the therapeutic blows which his attendant can muster, the response is not forthcoming. In this case the attendant's knowledge has been misdirected or perverted—he has "known too much". Apart, however, from these considerations which may be described as ethical or philosophical or indeed pertaining merely to common sense, the mundane question of cost should be mentioned. To the average patient this is important, for we all know that elaborate diagnostic procedures calling for special apparatus and trained specialist investigators ("Much Testing in the Lab", the B.B.C. might sing it!) are more costly than clinical examination in the consulting room or at the bedside with the careful use of eyes, ears and fingers; proprietary drugs in short supply are also dearer than drugs listed in the British Pharmacopœia. In these modern days when "life-saving drugs" are rightly supplied free of charge by the Government, the question of cost is apt to be overlooked, and unless the practitioner is watchful he may degenerate into a "steam-hammer prescriber".

Let us look at some of the "nut" problems in diagnosis and treatment. In diagnosis we may describe the problem as bound up with what has become known in these pages over the years as the worship of the test tube. Those who are given to this pastime prefer to use the elaborate and the expensive method perhaps because they think it will be quicker, or because they are lazy and want someone else to do the work for them, or because the elaborate makes more of a show and may impress the onlooker—the patient himself or some of his friends. There comes to mind the true story of an examinee who in the course of his clinical examination was investigating a patient's renal function and discussed with his examiners complicated chemical, biochemical and radiological methods but forgot the boiling test for albuminuria. Again we all know of the kind of investigator who has recourse to hystero-salpingography when the information sought can be obtained by the simple use of a uterine sound. And so on. But it is treatment on which most emphasis should be placed. The substances which are being abused more than any others are the sulphonamides and such drugs as streptomycin, "Chloromycetin" and aureomycin. The ordering of a sulphonamide in the presence of fever of any sort has with many practitioners become almost a conditioned reflex; the drug is often given in an empirical way and without any real attempt to discover the cause of the fever. Barbiturates, too, are distributed with largesse. They are added to so many prescriptions that one may be pardoned for wondering why—has the human

nervous system become generally unstable, or has a proper attempt been made to understand symptoms, or has such an attempt failed, or is a barbiturate added to an already complete, perhaps overburdened, prescription, just for full measure? When we come to the "life-saving" streptomycin, "Chloromycetin" and aureomycin, which are available free of charge to the patient under the provisions of *The Pharmaceutical Benefits Act*, the state of affairs is shocking. The quantities of these drugs ordered for individual patients by certain practitioners have been enormous. As a result some attempt has had to be made to control the distribution of the drugs. It is reported that large doses of penicillin have been used before the performance of such a straightforward operation as herniotomy. In the presence of actual or latent sepsis the administration of penicillin is justifiable and often necessary. To use it in the treatment of a healthy person before a set operation such as herniotomy is to imply that the patient has little or no natural resistance and that the surgeon need not be too careful in the washing of his hands. Of course we may see a reaction in which antibiotics will make surgical cleanliness old-fashioned. Perhaps that is what some of the new enthusiasts would like to see. It was said many centuries ago that men would be known by their works. This is true of medical practitioners who proclaim their ineptitude by the determined use of steam hammers for all the nuts that they have to crack.

Current Comment.

ORAL PENICILLIN THERAPY.

THE oral administration of penicillin, while known to be therapeutically effective in many cases, has not achieved wide use. For this fact the following reasons have been suggested by Paul Bunn *et alii*.[1] Firstly, the method was considered wasteful of an expensive material, because only from 20% to 33% of an orally administered dose of penicillin is absorbed from the intestinal tract. Secondly, the period for absorption is short, because the absorption of the drug is limited to a small segment of the gastro-intestinal tract, and this, coupled with rapid excretion, causes quite irregular or transitory demonstrable serum concentrations lasting only from two to four hours if small doses are employed. Thirdly, the presence of food in the gastro-intestinal tract interferes greatly with the efficient absorption of orally administered penicillin, with the inconvenient result that the drug has to take precedence over meals in the daily schedule. However, recent success with discontinuous therapeutic regimens and lessening in the cost of penicillin for oral administration suggested to Bunn and his colleagues that the whole matter warranted reconsideration. They carried out a series of investigations in which, first, they determined the serum concentrations of penicillin produced in normal or convalescent subjects after oral administration of the drug, and then they conducted a therapeutic test. Forty normal or convalescent hospital subjects were given 500,000 units orally in a variety of vehicles after an overnight fast, and serum concentrations of penicillin were determined periodically over an interval of eight hours. Twenty-six other similar subjects received 200,000 or 300,000 units and their blood levels were studied in the same way. In all cases therapeutically effective blood levels were obtained, and in approximately half of the patients receiving 500,000 units the concentration was still above 0·03 unit of penicillin per millilitre of serum at the end of eight hours. The concentrations were initially a good deal below those produced by the intramuscular injection of penicillin, but they decreased less rapidly and were the higher of the two towards the end of the eight-hour period. In the therapeutic trial a total of 104 patients with infections were treated. In 94 a satisfactory therapeutic response was observed after the administration of 500,000 units of penicillin twice or three times a day. In six others resolution of the infectious lesions was somewhat slower than expected, but complete cure eventually took place without change in therapy. In three of the remaining four cases it could be said that the oral treatment had failed, recourse being necessary to other forms of treatment. Bunn and his colleagues consider that regimens in which at least 500,000 units of penicillin are administered orally twice or three times a day are feasible in most cases in which penicillin is indicated. In certain conditions, such as bacterial endocarditis, meningitis and staphylococcal sepsis, in which high serum levels of penicillin are required or in which penicillin cannot easily penetrate into the involved area of inflammation, oral therapy is not advocated; but in the majority of cases of infections caused by micro-organisms susceptible to penicillin this form of treatment is safe, easy and reasonably inexpensive. When the drug is administered only twice or three times a day, scheduling of doses to avoid meals is no longer a problem. Thus all the original objections to oral penicillin therapy are in large measure overcome. The vehicle in which the penicillin is carried is unimportant, and no single vehicle has proved more efficacious than penicillin given in tap water. However, it is important that the administration of the tablet or fluid be so timed that the stomach is relatively empty. It should be noted that all patients in the series investigated had normally functioning gastro-intestinal tracts at the time when oral therapy was instituted. It is not advisable to administer penicillin by mouth to a patient with gastric dilatation or intestinal hypermotility. Absorption may be so impaired in either case that proper therapeutic response may not be obtained. The integrity of the gastro-intestinal tract is of great importance in the attainment of adequate blood concentrations of penicillin and the maintenance of therapeutic levels. In this series toxic reactions were noted in two patients, but the problem involved here does not appear to be greater than that associated with any other form of penicillin administration. Oral penicillin therapy has obvious advantages, and Bunn and his colleagues have presented a good case for the view that, used with discrimination, this form of therapy can be clinically quite satisfactory.

[1] *The Journal of the American Medical Association*, December 30, 1950.

THE CHANGING NATURE OF PNEUMONIA.

A PRACTITIONER who looks back on the medical wards of twenty-five years ago will remember most vividly perhaps those patients who suffered from acute lobar pneumonia. This disease, with its sudden onset, clear-cut symptoms and signs, and the dramatic, almost miraculous crisis which occurred in most cases, was of extraordinary interest. It was a battle to be fought by a triumvirate of patient, nurse and doctor against a swift and sudden foe. Nowadays, fortunately, lobar pneumonia in its classical form is rare. The question arises whether this is entirely due to the introduction of sulphonamides, penicillin and other antibiotics or whether there has been a change in the nature of the infection itself. H. A. Reimann[1] states that the incidence of pneumococcal lobar pneumonia is remarkably constant and has not been significantly disturbed either by the introduction of sulphonamides and antibiotics or by the transient pandemic of viral forms of pneumonia. The figures quoted in support of this statement come from such widely separated places as Philadelphia, Cincinnati, Scotland and New Zealand. The successful results of modern treatment undoubtedly give one the impression that acute lobar pneumonia occurs less often than formerly.

[1] *Annals of Internal Medicine*, November, 1950.

Reimann points out, however, that it was never a very common disease. He suggests that its striking characteristics, its high mortality rate in the past and its dangerous complications left lasting but exaggerated impressions of its frequency. Among the figures he quotes are those of Watson,[1] of New Zealand, who reports the occurrence at the Wellington Hospital of 1130 cases of acute lobar pneumonia in the years between 1928 and 1937, and 1087 between 1939 and 1948. According to these sets of collected figures, lobar pneumonia now affects an older age group and there seems to be an increase in the number of cases that are clinically "atypical".

There can, of course, be no doubt about the decline in the mortality rate of lobar pneumonia. It ranged from 20% to 40% in the days before the introduction of antiserum and sulphonamides, and now, since the introduction of penicillin, it varies from 5·7% to 9·3%. Even here the evidence is not quite clear-cut, for there has been a steady decline in the death rate of pneumonia in general since 1911, with an exception in 1918-1919 during the influenza pandemic. According to some authorities, the mortality rate of pneumonia in infants and in the aged is still high. In infants pneumonia is less easily recognized, and perhaps, therefore, less promptly treated. If some unforeseen catastrophe should stop the supply or distribution of modern therapeutic agents it would be interesting to see what would happen to the mortality rates for all ages.

Even if, as Reimann maintains, the incidence of acute pneumococcal pneumonia remains constant, changes in the character of pneumonia in general occur from time to time and seem to depend chiefly on the presence or absence of certain epidemic forms presumed to be caused by viruses. The rarer forms of the disease, such as staphylococcal or streptococcal pneumonia, may occur in small localized outbreaks. Vigilance is still necessary; it is important to establish the bacterial and/or viral cause of all forms of pneumonia, since different agents are sensitive to different antibiotics; and it is necessary to recognize epidemic forms of the disease. This may sound like a counsel of perfection, but at least an attempt should be made to follow it. Facilities for examination of sputum are often lacking, and diagnostic tests for rickettsial or viral infections are performed at only a very few Australian laboratories. Organization and extension of public health facilities in these regards are urgently needed before every case of pneumonia can be fully investigated.

AIR TRANSPORT OF PATIENTS.

IN a leading article in the issue of July 23, 1949, we discussed air travel and the medical fitness desirable for those who undertake it. Particular attention was drawn to an article by Whittingham, Barbour and Macgovern, appearing in the *British Medical Journal* of April 9, 1949. Another interesting and useful article has now appeared from America. Its authors are Colonel Benjamin A. Strickland, junior, and James A. Rafferty, from the Departments of Air Evacuation and Biometrics, United States Air Force School of Aviation Medicine,[2] and it is based on over 16,000 case reports on patients transported by air between January and October, 1949. Since June, 1948, all air transport of members of the armed forces of the United States, including the transport of patients, has been carried out by a single service, the Military Air Transport Service. Since August, 1949, air transport has been the sole method of moving patients for the armed forces. This is an interesting climax to a long and extensive experience of air transport, illustrated by the fact that the United States Air Force carried out nearly one and a half million patient-flights between 1942 and 1949. The large-scale investigation reported by Strickland and Rafferty appears to support this general policy, and they claim that their findings indicate that almost every patient suitable for transport by any means is suitable for transport by air. The 16,000 case reports were prepared on standard forms by graduate nurses who accompanied the patients in flight, and are stated to cover all patients carried by air in the period under review. The reports, completed at the end of each flight, list the diagnosis, symptoms occurring during flight, altitude and weather conditions, treatment necessary during flight, results of treatment and other relevant information. The only selection factors relate to two groups of patients not acceptable for air transport as a routine. The first group comprises those not normally acceptable, including those in such poor physical condition that the successful completion of their evacuation is doubtful, those with a fatal prognosis and in a moribund or semi-moribund state, those whose illnesses present a health menace to others in the aircraft or a public health menace (especially quarantinable diseases), those in a state of shock, those who have had attacks of coronary occlusion or *angina pectoris* within sixty days, and those with severe anæmia. The second group comprises those who need special consideration if they are carried and includes those with certain mental conditions, heart diseases, pneumothorax, severe asthma, recent severe internal hæmorrhage, recent intrathoracic or intraabdominal wounds or operations, certain skull fractures, recent severe head injuries or suspected intracranial injury, and maxillo-facial injuries. In actual practice very few patients have been rejected for air evacuation (for example, only five in a random sample of 2796 patients). The results suggest that air transport of patients is attended by far fewer symptoms than has been previously presumed, the incidence rate being only 7% for the entire series and the majority of these being apparently due, at least in part, to motion sickness. No patients died in flight or developed alarming symptoms requiring termination of the flight. Symptoms developing during flight were relieved by simple measures in 97% of cases. Analysis of experience with the second group of patients not carried as a routine—those who required special consideration, as flying might be expected to affect their clinical conditions—reveals a generally low incidence of symptoms, though the numbers in many cases were too small to be significant; symptoms were of a minor nature, and no untoward after-effects caused by air transport were reported. Conditions on the aircraft used were comparable with those on civilian aircraft, and to that extent the findings have a general application. No doubt the rejection rate for certain categories would be higher in the same-sized group of unselected civilians, but in general this very extensive experience supports the almost universal suitability of air transport for patients, especially when they are suitably prepared and occasionally special provision is made for their care in flight. It may be noted that none of the aircraft used had pressurized cabins and, as was pointed out by a speaker in the discussion that followed the reading of Strickland and Rafferty's paper, the types of aircraft discussed in the paper are relatively slow. We may well expect to see the incidence rate of symptoms reduced appreciably before long. Even under present conditions the principle of exclusive use of air evacuation for battle and other service casualties is established.

[1] *The New Zealand Medical Journal*, August, 1949.
[2] *The Journal of the American Medical Association*, January 20, 1951.

A GREAT AUSTRALIAN DIES.

A GREAT AUSTRALIAN, the Very Reverend John Flynn, D.D., O.B.E., has died. He was the father of the flying doctor services of Australia and more than any man earned the gratitude of the lonely men and women who live in the out-back. John Flynn was held in high honour by the people of Australia, and none were more alive to the value of his work than members of the medical profession. At sessions of the Australasian Medical Congress (British Medical Association) he held an honoured place for he was elected by the Federal Council as an honorary member. In the years to come when men and women who have to live in isolation continue to enjoy the advantages of medical care brought to them by "flying doctors", the name of John Flynn will be recalled with pride and gratitude. An account of his life and work will be published in a later issue.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Transplantation Technique for Paralysis of the Lateral Rectus Muscle.

CONRAD BERENS and LOUIS GIRARD (*American Journal of Ophthalmology*, July, 1950) describe the transplantation technique they employ for paralysis of the lateral rectus muscle and describe seven cases. They transplant the lateral halves of the superior and inferior recti and displace the nasal halves of these muscles temporally. This procedure is combined with recession of the medial rectus and resection of the lateral rectus. Tenon's capsule is transplanted with the muscle. In the seven cases reported there was improvement in abduction and the cosmetic result was excellent. The authors attribute the good result in part to the transplant of the medial halves of the vertical rectus muscles to the lateral side of their insertions, the recession of the medial rectus and the maintaining of Tenon's capsule intact.

Results of Operation for Cataract with Primary Glaucoma.

OTIS S. LEE and JACK E. WEIH (*Archives of Ophthalmology*, August, 1950) have analysed 100 cases in which cataract extractions were performed in eyes with primary glaucoma. The cases were divided into five groups. The first group of two cases was of chronic congestive glaucoma not controlled by miotics. An intracapsular extraction combined with peripheral iridectomy and anterior sclerectomy was performed in each case with good result. However, in these cases the authors recommend that the glaucoma be brought under control with miotics or surgery and that two months later the extraction be performed. In the two cases in which the combined operation was performed the cataract was mature and the fellow eye blind. The second group of 15 cases were of chronic non-congestive glaucoma not treated before cataract extraction. In all cases except one the tension was controlled without miotic therapy after the cataract extraction. The third group consisted of 15 cases of chronic non-congestive glaucoma in which the intraocular pressure was controlled by miotic therapy before cataract extraction. After extraction the pressure remained under control without further use of miotic in 12 cases. The type of iris surgery performed at the time of operation made little difference to the control of the tension. The fourth group consisted of 44 cases of chronic non-congestive glaucoma in which the intraocular pressure was controlled by glaucoma surgery prior to cataract extraction. The authors believe that the cataract extraction should not be performed until at least two months after the operation to control the glaucoma. Where a fistulizing operation has been performed the cataract should be removed intracapsularly. In four cases the glaucoma could not be controlled after cataract extraction, and in three of these cases extraction was extracapsular. The fifth group consisted of 24 cases of chronic non-congestive glaucoma with intraocular pressure uncontrolled by miotics or glaucoma surgery prior to cataract extraction. In these the authors recommend that some type of fistulizing operation be performed at the time of the cataract extraction.

Diabetic Retinopathy.

JONAS S. FRIEDENWALD (*American Journal of Ophthalmology*, August, 1950) reviews the progress of knowledge of diabetic retinopathy. He states that up to 1930 discussion was chiefly focused on the question of the relation of retinopathy to arteriosclerosis. In the pre-insulin era the weight of argument appeared to favour the disease as an arteriosclerotic manifestation, influenced in frequency and pattern by the diabetic process, but this had never been supported by histological evidence. From a study of serial sections of the retinal vascular trees in ten typical cases the author was able to conclude that retinopathy is not a consequence of local arteriosclerosis. Although atherosclerosis is responsible for the high incidence of coronary occlusion, of gangrene, and of cerebral vascular accidents in the diabetic, there would appear to be no direct relation between retinopathy and atherosclerosis. After the introduction of insulin, survival of juvenile diabetics became possible, and it was noted that diabetic retinopathy was a common complication in this group. From a survey of 2002 diabetics it was concluded that the most characteristic picture of diabetic retinopathy was punctate hæmorrhages and waxy exudates and that this is related to duration rather than severity of the diabetes. The frequency and progress of the retinopathy is little if at all influenced by diet or insulin. The average time between onset of diabetes and retinopathy is ten to fifteen years in juveniles and five to ten years in the older age group. In 25% of cases of retinopathy there is no ophthalmoscopic evidence of arteriosclerosis, and the frequency and severity of retinal arteriosclerosis are not significantly different in the diabetic with retinopathy from that in the non-diabetic of the same age. One feature of great importance is the tendency of the retinopathy to undergo periodical remissions and exacerbations. Patients with severe forms of diabetic retinopathy show increased capillary fragility. The author is not convinced that "Rutin" influences the course of diabetic retinopathy. Histological examination has shown that what were previously regarded as hæmorrhages are aneurysms, which are located most frequently at the point of capillary bifurcation, and that hæmorrhages and exudates are associated with the aneurysms. With the Hotchkiss-McManus staining technique it has been shown that there is a deposit of hyalin material in the capillary wall. This hyalin material appears to be identical with that which appears in the renal glomeruli in long-standing diabetes.

Toxoplasmosis.

ALBERT B. SABIN (*American Journal of Ophthalmology*, August, 1950) deals with the problems in toxoplasmosis which are of special interest in ophthalmological practice. He states that the commonest natural reservoir from which human beings may acquire toxoplasma is the dog and cat. The toxoplasma can come out in their fæces and urine. In addition, sheep, rabbits, guinea-pigs, voles and possibly squirrels must be considered. There is strong evidence to show that pigeons are also carriers. In discussing serological tests the author shows the limitation of the neutralization test in rabbit's skin and describes the more reliable quantitative in-vitro dye test, and mentions the new improved quantitatively controlled complement-fixation test. He studied 43 children who had chorioretinopathy and from this study was able to conclude that there exists a syndrome which clinically simulates healed congenital toxoplasmosis, but which is not due to toxoplasmic infection. If chorioretinopathy of presumably congenital origin is associated with cerebral calcification the chances are nine to one that serological tests will prove it to be due to congenital toxoplasmosis; on the other hand, if it is not associated with cerebral calcification, the chances are nine to one that it is not due to toxoplasmosis. When "healed" chorioretinopathy is first discovered in school-age children, the possibility of its being a healed, congenital toxoplasmic lesion can be entertained when the serum of both the mother and the child contains toxoplasmic antibodies. After the age of fifteen years the toxoplasmic antibodies in the normal population begin to rise very abruptly, and so it is more difficult to correlate chorioretinitis of unknown ætiology in adults with mere presence of such antibodies. In regard to therapy it has been shown that sulphathiazole administered to mice held infection in check, but there is no point in administering sulphonamides to a child with congenital toxoplasmosis as the damage has already been done.

Paracentesis and Ocular Ammonia Burns.

W. MORTON GRANT (*Archives of Ophthalmology*, September, 1950) conducted a series of experiments on rabbits' eyes which had been exposed for varying periods to ammonia. He found that repeated paracentesis produced no significant benefit in a series of 19 rabbit eyes treatment of which was begun one-half to forty-five minutes after exposure to ammonia of three degrees of severity. The pH of the aqueous humour was elevated after exposure to ammonia, but returned spontaneously to normal in fifteen to forty-five minutes and never reached damaging levels even with exposures which were devastating to the cornea. The author concludes that the efficacy of repeated paracentesis as a treatment for chemical burns of the eye is not supported by experimental evidence.

Intravenous "Novocain" Therapy in Ophthalmology.

ERWIN E. GROSSMAN *et alii* (*American Journal of Ophthalmology*, October, 1950) discuss the use of intravenous "Novocain" therapy in ocular conditions and report eight cases in which it was used. They state that when "Novocain" is given intravenously the drug accumulates at the site of the inflammation and exerts a definite selective action to break up the reflex pattern, and thereby relieves the vasospasm. Acute congestive glaucoma should respond favourably to intravenous "Novocain" therapy, with relief of pain and perhaps lowering of intraocular pressure. Some writers claim beneficial effects from the use of intravenous "Novocain" therapy in

branch obstructions of the central retinal vein. The technique of administration is to add one gramme of procaine hydrochloride to 1000 millilitres of isotonic saline solution, and to give this intravenously at a rate of 60 to 75 drops per minute; given at this rate the drug is analgesic. Over-stimulation of the central nervous system shown by convulsions is the most serious complication; it is controlled by cessation of administration and can be prevented to a great extent by the previous administration of a barbiturate.

Certain Hydrogenated Alkaloids of Ergot and Glaucoma.

ADOLPH POSNER (*American Journal of Ophthalmology*, October, 1950) has used hydrogenated alkaloids of ergot in 29 cases of glaucoma. He states that the drugs were without effect on chronic simple glaucoma, but that in the presence of pain and congestion, no matter what the cause of the tension, the intravenous administration of the drug resulted in almost immediate relief of pain and in a lowering of the ocular tension. The first one or two administrations are usually more efficacious than subsequent ones. In nearly all cases a dose of 0·3 milligramme is adequate for intravenous administration. It may be given at four-hourly intervals, but as a rule one or two injections a day are sufficient to control pain. The author considers that the drug is indicated in congestive glaucoma, either primary or secondary, to relieve pain, where miotics have been ineffectual, and in acute glaucoma preparatory to operation.

Oculo-Glandular Conjunctivitis Due to Leptothrix.

MARGARET HENRY (*The Journal of Pediatrics*, October, 1950) reports eight cases of Parinaud's oculo-glandular conjunctivitis. She states that this is a disease of the conjunctiva associated with enlarged preauricular and submaxillary lymph nodes and due to infection with the leptothrix. It is unilateral and occurs mainly in children with a history of exposure to cats. The constitutional symptoms are slight except for low-grade fever; mild eosinophilia is found. The conjunctival lesions appear as grey or yellowish necrotic areas beneath the epithelium. The conjunctiva is œdematous and congested, with secretion scanty or absent. Histologically the lesions are subepithelial areas of necrosis in which the predominant cell is the macrophage in various stages of necrosis. Irregular aggregations of very tiny unbranched filamentous microorganisms are present in the necrotic areas. The author recommends local and oral administration of sulphonamides, which she finds shortens the course of the disease considerably.

OTO-RHINO-LARYNGOLOGY.

Effect of Autonomic Denervation on Nasal Mucosa.

A. F. MILLONIG, H. E. HARRIS AND W. J. GARDINER (*Archives of Otolaryngology*, September, 1950) state that in the department of neurosurgery at the Cleveland Clinic Hospital various operative procedures have been carried out which interrupt the sympathetic or parasympathetic nerve supply to the head. These have afforded an opportunity of observing the effects on the nasal mucosa of such procedures. The parasympathetic fibres leave the brain stem as the *nervus intermedius*, forming a portion of the facial nerve. Some of these fibres pass via the greater superficial petrosal nerve to the spheno-palatine ganglion and thence to the mucous membrane of the nose and sinuses. The sympathetic outflow to the head has its cells of origin in the upper thoracic segments of the spinal cord. Preganglionic fibres pass by way of the anterior thoracic roots through the stellate ganglion mainly to the superior cervical ganglion, whence postganglionic fibres pass by way of the carotid plexus and deep petrosal nerve to the spheno-palatine ganglion and from there to the nasal mucosa. The parasympathetic fibres are generally vasodilator and secretomotor in function. Vasoconstrictor reactions are regulated through the sympathetic innervation. A series of 27 cases was available in which the sympathetic supply to the head was interrupted. Also there were six cases in which the parasympathetic supply to the nasal cavity was interrupted. In the cases in which the sympathetic nervous supply was interrupted, symptoms of nasal stuffiness and nasal discharge resulted. There was swelling of the nasal mucosa associated with increased secretion. These changes were the result of loss of vasoconstrictor tone and unrestricted parasympathetic activity. In the six cases in which the parasympathetic nerve supply was interrupted the mucous membranes were shrunken and dry.

Perceptive Deafness with Severe Neo-natal Jaundice.

NORMAN CRABTREE and JOHN GERRARD (*The Journal of Laryngology and Otology*, August, 1950) state that the term "kernicterus" is used to describe the neurological sequelæ in children who survive the deep infantile jaundice which is associated with Rh isoimmunization. Nuclear staining has been described in the brain of infants who die deeply jaundiced, and cellular changes have been recorded in the hippocampus and in the cochlear nuclei. Twenty subjects of kernicterus were subjected to a full otological examination, including pure tone audiometry, based upon conditioned responses when necessary. Sixteen of the 20 patients had perceptive deafness, and two were completely deaf. Three others had a conductive type of deafness. Only one of the patients tested appeared to have a normal range of hearing. In a number of cases the deafness was unsuspected until deliberately sought for. Opportunity arose to examine the brain stem and temporal bones of one patient who died while still jaundiced. Both organs of Corti, nerve fibres and ganglion cells were normal in appearance. In both the ventral and the dorsal cochlear nuclei in the brain stem, however, the cell bodies had completely disintegrated or faded away. In other instances the nucleus had disappeared. The authors conclude that perceptive deafness, usually of high-tone type, is a common finding in cases of kernicterus. The severity of the deafness bears no relationship to the severity of other disabilities. Attempts to assess intelligence in these cases may be misleading unless the otological findings are taken into account in selecting the types of test to be employed. In cases of perceptive deafness of obscure origin dating from childhood it is reasonable to ask if the child was severely jaundiced in infancy.

The Office Treatment of Chronic Otitis.

J. A. SULLIVAN and J. B. SMITH (*Annals of Otology, Rhinology and Laryngology*, June, 1950) state that gentle irrigation with 5% saline, aided by suction, is recommended as a method of cleansing the external auditory canal in most cases of chronic otitis. Care is necessary to avoid over-treatment and damage to the delicate integument of the canal. A 1% solution of aluminium acetate on a wick is recommended for reduction of œdema. Painting with a solution of 1% gentian violet and 95% alcohol every two days for a week is recommended for the diffuse form of inflammation and for the eczematous types. Ammoniated mercury in 2% dilution heals fissures and removes hyperkeratotic areas. The production of local sensitivity must be watched for, especially with penicillin, sulphonamides and streptomycin. For chronic *otitis media* thorough removal of debris with 5% saline, followed by suction and careful drying and then swabbing out with 95% alcohol, is recommended. Excess granulation tissue should be removed. The use of dyes, such as gentian violet, and "Zephiran" 1:1000 solution, as an antiseptic, has been found useful. It may be possible to remove a portion of cholesteatoma with suction, but recurrence is likely, and surgical treatment is often necessary owing to destruction of surrounding bone and development of symptoms indicating extension to adjacent parts.

Achalasia of the Crico-Pharyngeal Sphincter.

N. ASHERSON (*The Journal of Laryngology and Otology*, December, 1950) states that failure of the crico-pharyngeus to relax ahead of the oncoming bolus produces dysphagia which may be so slight as to be uncomplained of or the difficulty may be severe. The condition is not uncommon if sought for. The diagnosis rests on characteristic profile radiographs of the region. The normally constricted lower half of the hypopharynx remains permanently relaxed and patulous, giving the effect of marked elongation of the hypopharynx. Both valleculæ show out clearly when filled with opaque cream. A column of swallowed boli piles up in the hypopharynx, and this reservoir acts as a drip feed into the œsophagus. The lesions which produce this condition are complete pharyngeal paralysis, thyreotoxic myopathy involving the pharyngeal musculature, partial pharyngeal paralysis associated with left or right recurrent nerve paralysis, bilateral recurrent laryngeal nerve paralysis, and bulbar paralysis of the elderly. Achalasia differs from the condition found in complete paralysis of the œsophagus. Clinical examination shows the hypopharynx to be filled with frothy saliva. This accumulation may be sufficient to overflow into the larynx and cause a cough. A pharyngeal pouch or post-cricoid carcinoma may have to be excluded. In the Plummer-Vinson syndrome the profile X-ray pharyngogram is normal, while other signs are present to suggest the diagnosis.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the New South Wales Branch of the British Medical Association was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, on March 29, 1951, DR. G. C. HALLIDAY, the President, in the chair.

ANNUAL REPORT OF COUNCIL.

The annual report of the Council was received and adopted on the motion of Dr. H. R. R. Grieve, seconded by Dr. R. H. Macdonald. The report is as follows.

The Council presents the following report on the work of the Branch for the year ended March 29, 1951.

Membership.

The membership of the Branch is now 3125, as against 2930 at the date of the last report. The additions have included 237 elections, reelections and resumptions, and 75 removals into the area of the Branch; while the losses have included 8 by resignation, 64 removals out of the area of the Branch, 19 by default in payment of subscription, and 26 by death. The losses by death were as follows: Dr. W. K. Dale, Dr. E. J. Baxter, Dr. C. W. Sinclair, Dame Constance D'Arcy, D.B.E., Dr. H. L. Tooth, Dr. W. McDonnell Kelly, Dr. S. J. Blumer, Dr. A. T. Chapple, Dr. A. Lippmann, Dr. B. W. Stevenson, Dr. T. R. Pearce, Dr. M. M. Vernon, Dr. E. H. Molesworth, Dr. B. L. Middleton, Dr. Paul Tillett, Dr. Geoffrey Trahair, Dr. A. E. Panting, Dr. E. A. Woodward, M.C., Dr. H. J. West, Dr. V. H. Leigh-Barlow, Dr. G. W. Sinclair, Dr. T. P. Byrne, Dr. J. S. Harris, Dr. H. Lister, Dr. E. W. Byron, Dr. C. A. Hogg.

Meetings.

Ten ordinary meetings of the Branch (including the annual general meeting), one extraordinary meeting of the Branch, and ten clinical meetings were held; the average attendance was 110.

One ordinary meeting was held on April 27, 1950, in conjunction with a meeting of the Pharmaceutical Society of New South Wales, and eight in conjunction with meetings of special groups, namely: May 25, with the Section of Allergy and the Oto-Rhino-Laryngological Society of New South Wales; June 29, with the Section of Medicine and the Section of Neurology, Psychiatry and Neurosurgery; July 27, with the Section of Pathology and the Section of Medicine; August 31, with the Section of Obstetrics and Gynæcology and the Section of Pædiatrics; September 28, with the Section of Medicine and the Section of Pathology; October 26, with the Section of Pædiatrics and the Orthopædic Group (British Medical Association); November 30, with the Section of Neurology, Psychiatry and Neurosurgery; December 14, with the Section of Surgery.

The clinical meetings were held at the Rachel Forster Hospital for Women and Children, Royal Alexandra Hospital for Children, Royal Prince Alfred Hospital, Royal North Shore Hospital, Royal Hospital for Women, Lewisham Hospital, Sydney Hospital, Saint Vincent's Hospital, Saint George Hospital and Broughton Hall Psychiatric Clinic.

The business of the meetings included sixteen papers. At the extraordinary general meeting held on November 30, 1950, By-law 4 (Annual Subscription) was amended.

Representatives.

The Branch was represented of follows:

1. Council of the British Medical Association (1949-1952): Dr. Isaac Jones.
2. Representative Body of the British Medical Association (1950-1951): Dr. O. A. Diethelm.
3. Annual Meeting, British Medical Association, Liverpool, 1950: Delegate, Dr. O. A. Diethelm.
4. Federal Council of the British Medical Association in Australia: Dr. A. J. Collins, D.S.O., M.C., Dr. H. R. R. Grieve, Dr. W. F. Simmons, Dr. A. J. Murray, O.B.E.
5. Australasian Medical Publishing Company, Limited: Dr. W. F. Simmons, Dr. W. L. Calov, Professor L. E. Dods, M.V.O.
6. New South Wales Post-Graduate Committee in Medicine: Dr. A. C. Thomas, Dr. E. F. Thomson.
7. Ophthalmic Association, Limited: Dr. E. V. Waddy Pockley.
8. The Flying Doctor Service of Australia: Representative, Dr. George Bell, O.B.E.; Deputy Representative, Dr. J. G. Hunter.
9. Council of the Bush Nursing Association: Dr. G. C. Halliday.
10. Hospitals Contribution Fund of New South Wales: Dr. Hugh Hunter.
11. Saint John Ambulance Association: Dr. G. C. Halliday.
12. Standards Association of Australia: (i) Institutional Supplies Committee, Dr. S. W. G. Ratcliff; (ii) Sectional Committee on Interior Illumination of Buildings, Dr. N. M. Macindoe; (iii) Committee on Standards of Laboratory Glassware and Volumetric Glassware, Dr. F. S. Hansman; (iv) Committee on Protective Glass for Welding, Dr. N. M. Macindoe; (v) New South Wales Committee on Protective Occupational Clothing, Dr. W. T. Nelson; (vi) Paint and Varnish Sub-Committee No. 8, Dr. W. T. Nelson; (vii) New South Wales Committee on Eye Protection, Dr. N. M. Macindoe; (viii) Sectional Committee on Measuring Cups and Spoons, Dr. W. W. Ingram.
13. Medical Officers' Relief Fund (Federal): Local Committee of Management for New South Wales: Dr. E. H. M. Stephen, Dr. A. J. Murray, O.B.E., Dr. A. J. Collins, D.S.O., M.C.
14. Medical Appointments Advisory Committee (Hospitals Commission of New South Wales): Dr. L. A. Dey.
15. Special Departmental Committee for the Investigation of Maternal Deaths: Dr. E. A. Tivey.
16. Recreation and Leadership Movement: Professor Harvey Sutton.
17. Council of the Royal Society for the Welfare of Mothers and Babies: Sir Robert Wade, Dr. E. H. M. Stephen.
18. New South Wales Medical Board: Dr. J. R. Ryan.
19. Council of the New South Wales Institute of Hospital Almoners: Dr. R. A. R. Green.
20. Council of Education: Dr. A. J. Collins, D.S.O., M.C.
21. Examining Council of the Society of Laboratory Technicians of Australasia (New South Wales Branch): Dr. F. S. Hansman and Dr. E. F. Thomson.
22. Medical Finance, Limited, Board of Directors: Dr. E. A. Tivey, Dr. A. C. Thomas, Dr. George Bell, O.B.E., Dr. G. C. Halliday.
23. Council of the New South Wales Institute of Dietitians: Dr. H. R. R. Grieve.
24. Coordinating Council for the Physically Handicapped: Dr. R. A. R. Green.
25. Road Safety Council of New South Wales: Dr. G. C. Halliday; Committee for the Determination of Visual Standards for Motor Drivers, Dr. N. McA. Gregg; Committee for the Determination of Physical Fitness of Drivers of Motor Vehicles, Dr. J. H. Halliday; Committee to Examine and Report on matters dealing with Road Accidents involving Motor Cycles, Dr. I. D. Miller.
26. Federal Medical War Relief Fund: Local Committee of Management: Dr. A. J. Collins, D.S.O., M.C., Dr. A. C. Thomas, Dr. A. J. Murray, O.B.E.
27. Florence Nightingale Memorial Committee of Australia: Dr. B. T. Edye.
28. Australian Tuberculosis Association, New South Wales Division: Coordinating Committee: Dr. W. Cotter B. Harvey.
29. The Committee for Placement of Resident Medical Officers: Colonel A. M. McIntosh.
30. Australian Physiotherapy Association: Dr. B. G. Wade.
31. University of Sydney, Centenary Celebration Committee: Dr. G. C. Halliday.
32. The Cavalcade of Nursing Committee: Dr. H. R. R. Grieve.

Council.

(*a*) The attendance of members of the Council and of the standing committees was as set out in the accompanying table.

(*b*) The representatives of the Local Associations of Members appointed on the invitation of the Council to attend the regular quarterly meetings of the Council were as follows: Dr. W. McP. Roberts (Blue Mountains), Dr. L. S. Woods

(Border), Dr. J. B. Wilson (Broken Hill), Dr. J. M. Dowling (Brisbane Water District), Dr. L. Abramovich (Canterbury-Bankstown), Dr. G. N. M. Aitkens (Central Western), Dr. A. D. J. Frost (Eastern Suburbs), Dr. A. McNeil (Eastern District), Dr. J. F. Ireland (Far South Coast and Tablelands), Dr. F. P. M. Solling (Hunter Valley), Dr. G. W. Ashby (Illawarra Suburbs), Dr. H. S. Oag (Kuring-gai District), Dr. T. S. Douglas (Northern District), Dr. A. McL. Harper (South Eastern), Dr. E. S. Stuckey (Warringah District), Dr. W. W. Cameron (Western), Dr. R. F. Back (Western Suburbs).

Library.

Dr. J. Kempson Maddox was appointed to the position of Honorary Librarian.

Visitors to the library	6979
Books lent to members	1175
Journals lent to members	4396
Books added to the library	206
Journals added to the library	15

A comparison of the above figures with those of the previous year shows that the number of visitors to the library still continues to increase, the figure for this year being 455 more than for the 1949-1950 period, and there was also an increase from 236 to 262 in the number of inquiries from country members.

The total number of periodicals included in the library is 442, of which 266 are received currently, 139 being purchased by the Branch, and 127 received by donation.

The library accommodation is still proving inadequate, but it has not yet been possible to overcome this difficulty.

A copy of the library catalogue is now in the hands of the printers, and it is anticipated that it will be available for distribution in the very near future.

The Association would like to acknowledge its appreciation of the cooperation and assistance rendered throughout the year by the libraries of the other Branches and kindred institutions, and is pleased to record its grateful thanks for donations received from the following: the Editor, THE MEDICAL JOURNAL OF AUSTRALIA; Alfred Hospital, Melbourne; Australian Institute of Anatomy; Dr. S. P. Bellmaine; British Medical Association, Ceylon Branch; British Medical Association, Victorian Branch; Dr. H. L. Carruthers; Commonwealth Scientific and Industrial Research Organization; Food Preservation Department; Dr. W. S. Dawson; Dr. Wilfrid Evans; Dr. G. L. Ewan; Dr. J. P. Findlay; Dr. R. Hambridge; Dr. E. H. Hipsley; Dr. J. K. Maddox; Dr. F. A. Maguire; Dr. A. T. Nisbet; Dr. H. H. Nowland; Dr. K. C. O'Hanlon; Organization for Scientific Research, Indonesia; Dr. Alexander Owen; Miss M. M. Paton; Pharmaceutical Society of New South Wales; Public Health Department; Mrs. F. Savage; Dr. H. H. Schlink; Dr. Eva A. Shipton; Dr. R. J. Silverton; Dr. E. H. M. Stephen; Dr. A. C. Telfer; Dr. E. Trubert, Noumea; United Kingdom Information Service; United States Information Library; University of Queensland Post-Graduate Medical Education Committee; and the Section of Medicine; the Section of Neurology, Psychiatry and Neurosurgery; the Section of Obstetrics and Gynæcology; the Section of Pathology; the Section of Radiology; and the Oto-Rhino-Laryngological Society of New South Wales (British Medical Association).

Affiliated Local Associations of Members.

Blue Mountains (affiliated 1944): *Chairman*, Dr. L. Bamber; *Honorary Secretary*, Dr. N. Larkins. Membership 28. Four meetings were held.

Border (affiliated 1908): *Chairman*, Dr. R. A. Robertson; *Honorary Secretary*, Dr. F. G. Favaloro. Membership 16. Three meetings were held.

Brisbane Water District (affiliated 1948): *Chairman*, Dr. S. K. Dwyer; *Honorary Secretary*, Dr. J. M. Dowling. Membership 16. Eight meetings were held.

Broken Hill (affiliated 1942): *Chairman*, Dr. H. G. Hammond; *Honorary Secretary*, Dr. J. T. Cullen. Membership 10. Eleven meetings were held.

Canterbury-Bankstown (affiliated 1930): *Chairman* Dr. G. Russell; *Honorary Secretary*, Dr. J. T. St. Leger Moss. Membership 56. Five meetings were held.

Central Northern (affiliated 1910): *Chairman*, Dr. J. L. Deacon; *Honorary Secretary*, Dr. W. P. McLaughlin. Membership 83. Nine meetings were held.

Central Southern (affiliated 1909): *Chairman*, Dr. A. G. Wise; *Honorary Secretary*, Dr. J. P. Lyttle. Membership 43. Three meetings were held.

Central Western (affiliated 1910): *Chairman*, Dr. T. K. S. Whiting; *Honorary Secretary*, Dr. K. S. M. Brown. Membership 61. Four meetings were held.

Eastern District (affiliated 1913): *Chairman*, Dr. M. E. H. Elliott; *Honorary Secretary*, Dr. A. McNeil. Membership 32. Two meetings were held.

Eastern Suburbs (affiliated 1911): *Chairman*, Dr. A. D. J. Frost; *Honorary Secretary*, Dr. L. H. McMahon. Membership 129. Three meetings were held.

Far South Coast and Tablelands (affiliated 1935): *Chairman*, Dr. E. C. Blomfield; *Honorary Secretary*, Dr. J. F. Ireland. Membership 14. Three meetings were held.

Hunter Valley (affiliated 1947): *Chairman*, Dr. K. Klein; *Honorary Secretary*, Dr. F. P. M. Solling. Membership 45. Three meetings were held.

ATTENDANCE AT COUNCIL AND STANDING COMMITTEE MEETINGS.

	Council.	Committees.				
		Executive and Finance.	Organization and Science.	Medical Politics.	Hospitals.	Ethics.
ALEXANDER, M. S.	11	—	—	11	—	—
BELL, GEORGE, Honorary Treasurer	11	12	0	8	—	—
BLACKBURN, SIR CHARLES	9	—	—	—	—	—
BURNS, G. M.	10	—	3	—	—	—
DEAKIN, J. E. F.	11	—	—	11	—	—
DOUGLAS, T. S.	12	—	—	—	—	—
EDYE, B. T.	10	—	—	—	—	—
ELLIOTT, G. F.	8	—	—	10	—	—
GRIEVE, H. R. R., Honorary Secretary	12	8	0	9	—	—
HALLIDAY, G. C., President	12	11	2	12	—	—
HAMILTON, MARIE M.	8	—	1	—	—	—
HOWE, G. L.	12	10	—	10	—	—
MACDONALD, R. H.	12	10	—	—	—	—
MADDOX, J. K.,[1] Past President and Honorary Librarian	7	5	—	—	—	—
MAGUIRE, F. A.	4	—	—	—	—	—
MCINTOSH, A. M.[2]	10	—	—	—	—	—
MORROW, A. W.	10	—	4	—	—	—
MURRAY, A. J.	11	9	—	—	—	—
NELSON, T. Y.	10	—	—	12	—	—
RAWLE, K. C. T., President-Elect	9	3	2	8	—	—
SIMMONS, W. F.	12	12	—	12	—	—
THOMAS, A. C.	9	—	—	—	—	—
THOMSON, E. F.	12	—	4	—	—	—
WILLIS, H. H.	10	—	—	12	—	—
Meetings held	12	12	4	12	—	—

[1] Leave of absence from August to November.

[2] Full-time military service.

Illawarra Suburbs (affiliated 1913): *Chairman*, Dr. G. H. Gall; *Honorary Secretary*, Dr. G. W. Ashby. Membership 90. Seven meetings were held.

Kuring-gai District (affiliated 1929): *Chairman*, Dr. N. D. Barr; *Honorary Secretary*, Dr. J. Woolnough. Membership 73. Four meetings were held.

Northern District (affiliated 1911): *Chairman*, Dr. G. Archbold; *Honorary Secretary*, Dr. H. G. Royle. Membership 70. Three meetings were held.

North Eastern (affiliated 1913): *Chairman*, Dr. P. H. Doyle; *Honorary Secretary*, Dr. N. E. Brand. Membership 58. Five meetings were held.

Southern District (affiliated 1909): *Chairman*, Dr. A. C. Moran; *Honorary Secretary*, Dr. L. E. Goldsmith. Membership 49. Three meetings were held.

South Eastern (affiliated 1941): *Chairman*, Dr. J. B. Street: *Honorary Secretary*, Dr. M. C. McKinnon. Membership 39. Twelve meetings were held.

South Sydney (affiliated 1909): *Chairman*, Dr. L. E. Hewitt; *Honorary Secretary*, Dr. C. H. Jaede. Membership 45. Five meetings were held.

Warringah District (affiliated 1929): *Chairman*, Dr. G. M. B. Hales; *Honorary Secretary*, Dr. M. H. Elliot Smith. Membership 136. Six meetings were held.

Western (affiliated 1908): *Chairman*, Dr. A. W. McLaren; *Honorary Secretary*, Dr. S. R. Dawes. Membership 100. Four meetings were held.

Western Suburbs (affiliated 1908): *Vice Chairman*, Dr. J. O'Brien; *Honorary Secretary*, Dr. L. S. Hughes. Membership 130. Five meetings were held.

Annual Meeting of Delegates.

The thirty-seventh annual meeting of delegates of the affiliated local associations of members with the Council was held on Friday, September 22, 1950.

The delegates present at the meeting were as follows: Blue Mountains, Dr. W. M. Roberts; Border, Dr. L. S. Woods; Broken Hill, Dr. J. T. Cullen; Brisbane Water District, Dr. J. M. Dowling; Canterbury-Bankstown, Dr. L. Abramovich; Central Southern, Dr. J. P. Lyttle; Central Northern, Dr. L. N. Ferrari; Central Western, Dr. G. N. M. Aitkens; Eastern Suburbs, Dr. L. H. McMahon; Eastern District, Dr. A. McNeil; Far South Coast and Tablelands, Dr. J. F. Ireland; Hunter Valley, Dr. F. P. M. Solling; Illawarra Suburbs, Dr. G. W. Ashby; Kuring-gai District, Dr. H. S. Oag; Northern District, Dr. R. J. Jackson; North Eastern, Dr. J. L. Roberts; Southern District, Dr. G. Mackintosh; South Eastern, Dr. A. McL. Harper; South Sydney, Dr. C. H. Jaede; Warringah District, Dr. E. S. Stuckey; Western, Dr. R. D. Mulvey; Western Suburbs, Dr. S. Lackey.

Special Groups for the Study of Special Branches of Medical Knowledge.

Allergy (inaugurated 1947): *Chairman*, Dr. R. S. Steel; *Honorary Secretary*, Dr. Bernard Riley. Membership 9. Four meetings were held, one in conjunction with a meeting of the Branch.

Anæsthesia (inaugurated 1934): *Chairman*, Dr. J. F. McCulloch; *Honorary Secretary*, Dr. L. T. Shea. Membership 36. Seven meetings were held.

Medicine (inaugurated 1924): *Chairman*, Dr. K. B. Noad; *Honorary Secretary*, Dr. Selwyn Nelson. Membership 50. Seven meetings were held, three in conjunction with meetings of the Branch.

Neurology, Psychiatry and Neurosurgery (inaugurated 1924): *Chairman*, Dr. G. Phillips; *Honorary Secretary*, Dr. F. J. Scanlan. Membership 85. Four meetings were held, two in conjunction with meetings of the Branch.

Obstetrics and Gynæcology (inaugurated 1925): *Chairman*, Dr. G. G. L. Stening; *Honorary Secretary*, Dr. F. N. Chenhall. Membership 105. Five meetings were held, one in conjunction with a meeting of the Branch.

Orthopædic Group (British Medical Association) (inaugurated 1923): *Chairman*, Dr. Hugh Barry; *Honorary Secretary*, Dr. W. S. L. Stening. Membership 22. Eight meetings were held, one in conjunction with a meeting of the Branch.

Oto-Rhino-Laryngological Society of New South Wales (inaugurated 1924): *Chairman*, Dr. B. B. Blomfield; *Honorary Secretary*, Dr. A. M. Bryson. Membership 46. Six meetings were held, one in conjunction with a meeting of the Branch.

Pædiatrics (inaugurated 1924): *Chairman*, Dr. R. A. R. Green; *Honorary Secretary*, Dr. D. G. Hamilton. Membership 64. Seven meetings were held, two in conjunction with meetings of the Branch.

Pathology (inaugurated 1924): *Chairman*, Dr. G. V. Rudd; *Honorary Secretary*, Dr. R. D. K. Reye. Membership 73. Seven meetings were held, two in conjunction with meetings of the Branch.

Radiology (inaugurated 1926): *Chairman*, Dr. J. A. Vote; *Honorary Secretary*, Dr. E. W. Frecker. Membership 83. Seven meetings were held.

Surgery (inaugurated 1925): One meeting was held in conjunction with a meeting of the Branch.

Urology (inaugurated 1940): *Chairman*, Dr. K. Kirkland; *Honorary Secretary*, Dr. H. Cummine. Membership 13. Three meetings were held.

By-Laws.

At an extraordinary general meeting of the association held on November 30, 1950, By-law 4 was amended to provide that a member engaged in practice solely as an employee of any Authority, Corporation, Company, Association or Body shall pay a subscription of six pounds six shillings, instead of eight pounds eight shillings. The By-law was also amended to provide that in the case where a member paying a subscription at a particular rate becomes entitled under the By-laws to have his subscription reduced to another rate, the reduction shall apply only from the first day of January next after he shall have given notice to the association of the happening of the event or the change in his position which entitles him to have his subscription reduced, and until that date he shall continue to pay his subscription at the rate which would be payable by him if such event had not happened or his position had not changed.

The Federal Council of the British Medical Association in Australia.

Dr. A. J. Collins.

Dr. A. J. Collins, Vice-President of the New South Wales Branch and a representative of the Branch on the Federal Council for a number of years, was appointed President of the Federal Council at its meeting held in Melbourne from February 26 to 28, 1951.

Meetings.

The Federal Council of the British Medical Association in Australia met in Brisbane on May 26, 27, 28, 29, 30 and 31, 1950, in Sydney on September 23 and 24, 1950, and in Melbourne on February 26, 27 and 28, 1951.

At these meetings the Branch was represented by Dr. A. J. Collins, Dr. H. R. R. Grieve, Dr. W. F. Simmons and Dr. A. J. Murray.

British Commonwealth Medical Conference.

Dr. H. R. R. Grieve, Honorary Secretary, and Dr. A. E. Lee, a member of the Queensland Branch, represented the Federal Council of the British Medical Association in Australia at the Second Annual Meeting of the British Commonwealth Medical Conference held at Brisbane on May 23, 24 and 25, 1950.

Dr. J. G. Hunter, Medical Secretary, acted as the Local Honorary Secretary.

The Committee for Placement of Resident Medical Officers.

The Committee for the Placement of Resident Medical Officers was appointed by the Minister for Health for New South Wales following an interview which representatives of the British Medical Association, New South Wales Branch, and the New South Wales Post-Graduate Committee in Medicine had with him on October 7, 1948. At this interview the representatives of these two organizations stressed the difficulties which would be experienced in finding vacancies for resident medical officers in the years 1951, 1952 and 1953 as a result of the large number of students who would be graduating.

The committee, consisting of Dr. A. B. Lilley, Chairman, Hospitals Commission of New South Wales, Colonel A. M. McIntosh representing the British Medical Association, New South Wales Branch, Dr. George Bell representing the New South Wales Post-Graduate Committee in Medicine, and Professor Bruce T. Mayes representing Professor H. R.

Dew, Dean of the Faculty of Medicine, met following the publication of the results of the Final Degree Examination in Medicine for the purpose of allocating the successful candidates to hospital appointments other than those made by the conjoint boards of teaching hospitals. Prior to the meeting, an appeal had been made to the various hospitals by the Chairman, Hospitals Commission of New South Wales, to increase the number of their resident medical officers, the appeal meeting with a great deal of success.

The committee reported that it was able to place all successful graduates desiring appointments in New South Wales.

Reports indicate that the work of the committee was greatly appreciated.

The help of the Secretariat of the British Medical Association, New South Wales Branch, was made available to the committee in its work.

Friendly Society Contract Practice.

Termination of the Common Form of Agreement.

A plebiscite taken in September, 1950, indicated that the large majority of members engaged in lodge contract practice desired to terminate the capitation method of payment of medical practitioners engaged in friendly society contract practice and substitute in lieu thereof a fee for service method of payment. This plebiscite was taken after the matter had been fully discussed at special meetings of delegates of affiliated local associations with Council held on April 1, 1950, and May 13, 1950.

The fee for service method of payment is the policy of the Federal Council. It will come into operation in all the States.

In order to allow of satisfactory arrangements being made by the friendly societies with the Government, the Friendly Societies Association of New South Wales was informed that the Council was prepared to recommend to members that they continue to provide medical services to subvention members until June 30, 1951, the rates for such attendance to be in accordance with the Council's interpretation of Clause 13 of the Common Form of Agreement, viz:

	Metropolitan (per annum).	Country (per annum).
	£ s. d.	£ s. d.
Male	2 5 6	2 15 7
Single female ..	1 14 1	2 1 8

This offer does not apply to subvention members who are pensioners and who will receive their medical services through the Government scheme.

Clause 13.

Owing to a difference of opinion between the Friendly Societies Association of New South Wales and the Council as to the interpretation of Clause 13 of the Common Form of Agreement, it was agreed by both parties that a case should be presented to Court for an interpretation. A writ was accordingly issued in the name of a member who agreed to act as nominal plaintiff. However, in view of the decision to terminate the Common Form of Agreement on March 31, 1951, it was decided that no good purpose would be served by proceeding with the case and accordingly the writ was withdrawn.

Industrial Contract Practice.

Following a meeting of representatives of members practising in the industrial areas of the South Eastern Medical Association, Hunter Valley Medical Association, Central Northern Medical Association and the Blue Mountains Medical Association, with representatives of Council, the Council decided that the following rates should apply in industrial contract practice:

(*a*) Complete service (of a general practitioner nature) including confinements and operations, but excluding Workers' Compensation cases, dental anæsthetics, week-end and night calls and supply of medicine. 5s. per fortnight, and in the case of single females, 3s. per fortnight.

(*b*) Complete service (of a general practitioner nature) as in (*a*), but excluding confinements. 4s. 6d. per fortnight, and in the case of single females, 3s. per fortnight.

(*c*) Incomplete service (of a general practitioner nature) in which the services are similar to those under the Friendly Society Common Form of Agreement. Ordinary members 3s 6d. per fortnight, pensioners 3s. per fortnight, single females and widows without dependants 1s. 9d. per fortnight.

The above rates are subject to variations in the basic wage, that in existence on January 1, 1951, being the amount on which amendments to the capitation rate are to be determined.

For after-hour calls from 6 p.m. to 8 a.m. and from 12 noon Saturday to 8 a.m. Monday and on public holidays a fee of 10s. per attendance may be charged.

Mileage charges are 6s. per mile after the first two miles one way in the daytime and 7s. 6d. per mile after the first two miles one way in the night time.

The above terms are now in operation with minor amendments in the various industrial areas mentioned.

With the object of discussing with representatives of organizations concerned the rates and conditions applicable to the service to be rendered by members practising in mining districts, the Council was represented at conferences as follows:

Wollongong, November 26, 1950, Dr. G. C. Halliday (President), Dr. H. R. R. Grieve, Dr. W. F. Simmons, Dr. Hugh Hunter (Assistant Medical Secretary). Cessnock, January 28, 1951, Dr. G. C. Halliday (President), Dr. H. R. R. Grieve, Dr. W. F. Simmons, Dr. J. G. Hunter (Medical Secretary). Broken Hill, February 10 and 11, 1951, Dr. G. C. Halliday (President), Dr. J. G. Hunter (Medical Secretary).

Fees Payable to Legally Qualified Medical Practitioners for Services Rendered on Behalf of the Government on Request.

Some two years ago, representations were made by the Council to the Minister for Health for an increase in the fees payable to legally qualified medical practitioners for services rendered on behalf of the Government on request. These fees were determined by regulations promulgated on September 9, 1909.

New regulations have now been promulgated increasing the fees, but whilst there has been a general increase in the fees, the amounts recommended by Council have not been accepted. The matter is receiving the further consideration of Council.

Cortisone: Appointment of a Committee to Advise on its Uses in Public Hospitals.

At the request of Dr. A. B. Lilley, Chairman, Hospitals Commission of New South Wales, a committee was appointed by Council to advise the Hospitals Commission in regard to the suitability or otherwise of patients for treatment by cortisone in public hospitals. This request was made by the Chairman, Hospitals Commission of New South Wales, in view of the very limited supply of this drug and the demand being made by hospitals for its supply.

The committee appointed consists of Dr. A. J. Collins, Chairman, Dr. H. R. R. Grieve, representing general practitioners, Dr. J. K. Maddox, Royal Prince Alfred Hospital, Dr. W. L. Calov, Sydney Hospital, Dr. I. A. Brodziak, Royal North Shore Hospital, and Dr. R. J. Taylor, Saint Vincent's Hospital.

Indiscriminate Use of Benzedrine.

The attention of the Director-General of Public Health was drawn to the indiscriminate manner in which benzedrine, in its various forms, is being used by the public. The Council considered that this was not in the public interest, and recommended that the sale of benzedrine should be controlled by having it classified as a dangerous drug.

A reply from the Department of Health indicated that the powers prescribed under the *Police Offences (Amendment) Act* relating to dangerous drugs were strictly limited, and it was not considered possible to include benzedrine on the list. The Department stated, however, that the matter would be further considered in connexion with a proposal to amend the existing *Poisons Act*, with a view to making suitable provision for the control of drugs of this nature, when an amendment of such Act is being dealt with by Parliament.

Subsidized Practices.

Following further representations to the Minister for Health, there has been another increase in the remuneration payable in those practices in sparsely populated areas in which medical practitioners are providing full-time medical services on a subsidized basis. Whereas previously the guaranteed income was £1200 *per annum*, it is now £1500 *per annum*.

The Council also made a recommendation to the Minister for Health that the mileage subsidy rate in those cases where medical services in sparsely populated areas are provided by medical practitioners from neighbouring towns, should be increased from 1s. to 1s. 6d. per mile. The Minister did not accept the recommendation of Council, but increased the rate to 1s. 3d. per mile.

Bus Termini in Macquarie Street.

The establishment of bus termini in Macquarie Street created a problem in the way of noise, brought about by the stopping and starting of buses. This has rendered it difficult for many practitioners to carry out their work efficiently.

The matter was taken up with Mr. R. Winsor, Director of Transport and Highways, who expressed appreciation of the problem submitted to him. By a rearrangement of the sites originally determined for the termini, a fraction of the noise was abated.

The position being still very acute, further representations were made in the matter, but the Council was advised that the position could not be altered. The situation at present is very unsatisfactory, many complaints being received from members of the increasing noise and a consequent interference with their professional work.

British Medical Association Food Parcels Fund.

With the closing down of the Food for Britain Appeal, the Council decided, with a great deal of reluctance, not to issue any further appeal to members to subscribe to the British Medical Association Food Parcels Fund.

Numerous letters of appreciation were received from the beneficiaries of the Royal Medical Benevolent Fund, who were recipients of the parcels. The appreciation of the Royal Medical Benevolent Fund was expressed in a letter from Lord Webb-Johnson, which appeared in the issue of THE MEDICAL JOURNAL OF AUSTRALIA of November 4, 1950.

The Council wishes to express its thanks to all those members who, through their generosity, brought so much happiness into the lives of some of our less fortunate colleagues and their dependants in Great Britain.

The total amount subscribed to the Fund was £1555 18s.

Department of Medical Sociology and Research.

The department has continued its work of popular education, mainly through broadcasting, including short commentaries on items of medical news. As before, assistance has been given to the Press in its use of medical news and articles.

During the year more than 200 talks were prepared for delivery by the association's spokesman over the National Stations. The talks previously given in three of the Commission's programmes were continued, and a new feature has been introduced. This is a weekly talk in the session "Where Old Friends Meet", intended for listeners of the age group of sixty-five years and older. To date 48 such talks have been given, on many aspects of gerontology and the general welfare of persons of the higher ages.

Some talks have been quoted from or reproduced in the *A.B.C. Weekly*. The association has received (December 19, last) the Broadcasting Commission's thanks for "continuous cooperation" in this work.

NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

Balance Sheet as at December 31, 1950.

	£ s. d.	£ s. d.
FIXED LIABILITIES.		
Debentures—		
62—4% Series "A" at £10 each	620 0 0	
350—4·65% Series "B" at 50 each	17,500 0 0	
210—4·65% Series "C" at £10 each	2,100 0 0	
	20,220 0 0	
Less Amount Unpaid	54 0 0	
	20,166 0 0	
General Reserve Fund (used in the business)	1,250 0 0	
Australian Mutual Provident Society (secured by mortgage over property, B.M.A. House)	36,000 0 0	
		57,416 0 0
CURRENT LIABILITIES.		
Sundry Creditors	1,121 9 2	
Interest Accrued on Mortgage	127 10 0	
Deposit at Call	700 0 0	
Commercial Banking Company of Sydney, Limited—Premises and Branch Current Accounts	856 3 2	
		2,805 2 4
OTHER CREDIT BALANCES.		
Subscriptions Paid in Advance	98 9 2	
Provision for Taxation	1,443 0 0	
Provision for Painting Building	800 0 0	
Superannuation Fund Reserve (as *per contra*)	871 11 10	
		3,213 1 0
ACCUMULATED FUNDS.		
Balance at December 31, 1949	95,348 17 8	
Add Surplus for year ended December 31, 1950—		
Branch Surplus £3,622 10 5		
Less Premises—		
Deficit £132 9 7		
	3,490 0 10	
		98,838 18 6
		£162,273 1 10

	£ s. d.	£ s. d.
FIXED ASSETS.		
Land and Buildings, B.M.A. House —at Cost *less* Depreciation on Building	141,640 11 3	
Library—at Valuation *less* Depreciation	4,353 1 11	
Office Furniture and Equipment—at Valuation *less* Depreciation	1,399 18 9	
Debentures—Australasian Medical Publishing Company, Limited	4,400 0 0	
Deposit on Debentures — Australasian Medical Publishing Company, Limited	146 10 4	
Commonwealth Treasury Bonds (Face Value)	7,000 0 0	
		158,948 2 3
FLOATING ASSETS.		
Sundry Debtors (after making provision for doubtful debts)—		
Sundry Tenants, Rents *et cetera*	1,609 17 7	
Cash on Hand	12 0 9	
		1,621 18 4
OTHER DEBIT BALANCES.		
Prepaid Insurances, Rates *et cetera*		831 9 5
Superannuation Fund Investments Account (as *per contra*)		871 11 10
		£162,273 1 10

Sydney, February 19, 1951.

G. C. HALLIDAY, President.
GEORGE BELL, Honorary Treasurer.
R. J. STIFFE, Financial Secretary.

We have examined the foregoing Balance Sheet with the Books of Account of the New South Wales Branch of the British Medical Association, and, having obtained all the information and explanations we have required, we are of the opinion that such Balance Sheet is properly drawn up so as to exhibit a true and correct view of the state of the Company's affairs according to the best of our information and the explanations given to us and as shown by the Books of the Company. In our opinion the Register of Members and other records which the Company is required to keep by the New South Wales Companies Act, 1936, or by its Articles have been properly kept.

Sydney.

F. W. DUESBURY AND COMPANY,
Chartered Accountants (Aust.).

PREMISES ACCOUNT.

Income and Expenditure Account for the Year ended December 31, 1950.

	£ s. d.	£ s. d.		£ s. d.
To Wages	4,110 8 0		By Rent Revenue	18,357 14 0
„ Salaries	1,119 10 11		„ Interest—Treasury Bonds	16 5 0
„ Water and Sewerage Rates	1,047 8 10		„ Balance, being Deficit for year ended December 31, 1950, transferred from Accumulated Funds Account	132 9 7
„ Municipal Rates	674 15 0			
„ Insurance	264 10 0			
„ Maintenance—				
Building	1,044 7 4			
Lifts	713 7 9			
Central Heating	378 1 0			
„ Federal Land Tax	131 1 3			
„ Electricity and Gas	541 7 5			
„ Telephone	55 15 6			
„ Audit Fee	42 0 0			
„ Legal Expenses	5 7 6			
„ Stamps and Stationery	2 16 11			
„ Stamp Duty Receipt Books	4 19 10			
„ Architect's Report *re* Waterproofing Building	26 5 0			
„ Exchange and Bank Charges	6 0 0			
„ Costs *re* Rent Application	236 5 0			
„ Travelling Expenses	2 13 0			
„ Donations	105 0 0			
„ Sundry Expenses	27 7 6			
„ Pay Roll Tax	118 13 9			
		10,658 1 6		
„ Interest—				
Commercial Banking Company of Sydney, Limited	7 0 9			
A.M.P. Society	1,630 6 8			
Debentures	933 16 5			
Deposit	35 0 0			
		2,606 3 10		
„ Allowance for Depreciation of—				
Building	2,800 0 0			
Plant	7 9 3			
„ Provision for Painting of Building	800 0 0			
		3,607 9 3		
„ Provision for Taxation		1,634 14 0		
		£18,506 8 7		£18,506 8 7

British Medical Agency of New South Wales, Limited.

The annual general meeting of the British Medical Agency of New South Wales, Limited, was held on October 3, 1950.

The report of the directors, presented by the chairman, Dr. George Bell, showed an improvement in the profit for the year ended June 30, 1950.

The directors look forward to the continued support of their own agency by members of the profession.

Medical Finance, Limited.

The annual meeting of Medical Finance, Limited, was held on October 3, 1950.

The chairman, Dr. George Bell, presented the report of the directors for the year ended June 30, 1950. The report showed that a small profit had been made. The profit is limited because there is little demand for the company's funds, prospective borrowers being accommodated at lower and more attractive interest rates through other channels.

Premises Revenue Account.

The Premises Revenue Account discloses a net deficit of £132 as against a net surplus of £1167 for the year ended December 31, 1950, thus showing a decrease of £1299 in the net surplus revenue earned. This decrease is accounted for by a net increase in expenditure of £1318 and a net increase in income of £19 as set out in detail on the accompanying comparative statement.

A comparison of percentages of expenditure to rent revenue with those at December 31, 1949, is as follows:

	1949.	1950.
Percentages of expenses to revenue	93·7%	100·8%
Percentage of surplus to revenue	6·3%	—
	100·0%	—

The percentage of rent revenue, expenses and depreciation and the percentage of net surplus for the year to the capital value of the land and building (British Medical Association House) as shown by the books at December 31, 1950, namely, £141,648, with the previous year's percentages in parentheses, are as follows:

Rent Revenue (including amount charged for British Medical Association Branch Offices, *et cetera*)	12·9%	(12·7%)
Sundry Expenses, Interest and Provision for Painting Building	11·1%	(10·0%)
Depreciation of Building	1·9%	
	13·0%	(1·9%)
Net surplus for year	−0·1%	(0·8%)

Financial Statement.

The Council has pleasure in presenting to members the balance sheet and accounts in respect of the financial year which terminated on December 31, 1950.

The net surplus of revenue over expenditure for the year amounted to £3490 0s. 10d. after making provision for all known expenditure.

The sum of £3268 16s. 7d. has been written off for depreciation of the building (British Medical Association House), plant, office furniture and equipment and the library.

The sum of £800 has been provided out of the current year's revenue to create a reserve for painting the exterior of the building. This amount for the time being is used in the business of the association.

G. C. HALLIDAY,
President.

The balance sheet of the Branch and the income and expenditure account of the Branch and of the premises were received and adopted on the motion of Dr. George Bell, seconded by Dr. W. F. Simmons.

ELECTION OF OFFICE BEARERS.

Dr. G. C. Halliday announced that the following had been elected members of the Council for the ensuing year.

Elected as Representing the General Body of Members.—Dr. M. S. Alexander, Dr. George Bell, Sir Charles Blackburn,

Dr. J. E. F. Deakin, Dr. B. T. Edye, Dr. H. R. R. Grieve, Dr. A. S. Johnson, Dr. R. H. Macdonald, Dr. A. M. McIntosh, Dr. F. A. Maguire, Dr. A. W. Morrow, Dr. A. J. Murray, Dr. T. Y. Nelson, Dr. W. F. Simmons, Dr. A. C. Thomas, Dr. E. F. Thomson.

Elected as Representing Women Members.—Dr. Phyllis M. Anderson.

Elected as Representing the Public "Government" Medical Services.—Dr. H. H. Willis.

Elected as Representing Country Local Associations.—Dr. B. A. Cook (one yet to be elected).

Elected as Representing Metropolitan Local Associations.—Dr. G. F. Elliott, Dr. G. L. Howe.

Messrs. F. W. Duesbury and Company were appointed auditors for the ensuing year.

Election of Representatives of the Branch at the Annual Representative Meeting of the British Medical Association, London.

It was resolved on the motion of Dr. T. Y. Nelson, seconded by Dr. H. H. Willis, that Dr. E. F. Thomson should be appointed the representative of the New South Wales Branch to attend the Annual Representative Meeting of the British Medical Association, 1951, to be held at London on June 13 to 16, 1951.

Incoming President's Address.

Dr. K. T. C. Rawle delivered his incoming president's address (see page 709).

Induction of President.

Dr. G. C. Halliday inducted the President for the year 1951-1952, Dr. K. C. T. Rawle. Dr. Rawle thanked the members for his election.

Correspondence.

NITROUS OXIDE–THIOPENTONE–CURARE ANÆSTHESIA: A PRELIMINARY REPORT ON AN APPARENTLY SATISFACTORY TECHNIQUE.

SIR: I am grateful to Dr. W. H. J. Cole for drawing attention (THE MEDICAL JOURNAL OF AUSTRALIA, April 21, which I have just received) to a detail of technique which was omitted from my article through considerations of space. One of the sections which could not be published was entitled "Some Practical Details", and contained the following relevant passage:

> Satisfactory results are obtained if one sets the flow-rate at 0·5 litre per minute for each gas from the commencement. My own gas analyses appear to indicate that the oxygen content settles at around 30% fairly soon, at least within fifteen or twenty minutes. However, in view of certain reports (Crowley *et alii*, 1948) on times taken to achieve stable gas mixtures with small flow-rates, I sometimes practise a rapid "continuous flow" technique for the first ten minutes or so. With more rapid elimination of nitrogen and earlier establishment of equilibrium, it is possible that one may thus diminish still further the requirements of TPC solution.

While agreeing entirely with Dr. Cole's statement, I would say that I do not now feel it matters much for practical purposes whether one observes this technical consideration or not.

Might I have your indulgence to mention briefly on this occasion several other points which were similarly omitted in the published article?

1. To date, I have given nearly 700 anæsthetics by this technique, with closely similar results to those of my recorded series, and with an equal degree of satisfaction.

2. The method is now used routinely in two classes of patient previously regarded *sub judice*, namely, women undergoing Cæsarean section and children old enough for closed-circuit methods.

3. Recently there has been published (Burstein *et alii*, 1950) an experimental confirmation of what had become a "clinical impression" in my recorded series. Various unwelcome autonomic reflexes which can occur during anæsthesia (vagal and carotid sinus reflexes and the like) have been found experimentally to be inhibited or depressed by adequate doses of curare. This may be a most important discovery, and one which suggests yet another advantage in the nitrous oxide-TPC technique.

4. Post-operative vomiting, following any technique, could conceivably have an important basis in gastro-intestinal hypotonia associated as a matter of course with surgical procedures under general anæsthesia (perhaps arising out of defective cholinergic activity at the level of Auerbach's plexus). Hence the importance of withholding oral fluids in the immediate post-anæsthetic period.

5. I prepare TPC solution in an arrangement of sterile tubing, three-way taps *et cetera*, from such packings as "Soluvac" flasks of distilled water, five-gramme bottles of "Intraval" powder and two-millilitre ampoules of *d*-tubocurarine chloride. No discernibly adverse results have followed the use of solutions five or six days after preparation, although solutions are rarely employed which are more than three days old. *In vitro*, I have found no visible change in solutions after several weeks' storage.

6. The cost of TPC solution in my own practice ranges from 11s. 9d. to 12s. 6d. for 40 millilitres (depending on whether bulk packings are employed).

7. In the absence of untoward complications, increased pulse rates may be due to lightening anæsthesia or (temporarily and in certain patients) to successive injections of TPC solution itself.

8. The more valuable sign-posts of depth in the nitrous oxide-TPC technique appear to be (*a*) voluntary or reflex movement—a slight movement of hand or foot or vocal cords may precede other indications, (*b*) alterations in pulse rate, respiration rate and blood pressure, (*c*) adequacy or otherwise of abdominal relaxation. Pending the arrival of another Guedel, personal experience probably remains our best teacher in this field.

Yours, etc.,
RONALD V. PRATT

12 Grange Street,
Swanbourne, Western Australia.
April 28, 1951.

References.

Crowley, J. H., Faulconer, A., junior, and Lundy, J. S. (1948), "Certain Factors Influencing the Percentage of Oxygen in Mixtures of Nitrous Oxide and Oxygen", *Current Researches in Anesthesia and Analgesia*, Volume XXVII, page 255.

Burstein, C. L., Jackson, A., Bishop, H. F., and Rovenstine, E. A. (1950), "Curare in the Management of Autonomic Reflexes", *Anesthesiology*, Volume XI, page 409.

BROAD VIEWS ON SEX-WORSHIP.

SIR: As your reviewer (THE MEDICAL JOURNAL OF AUSTRALIA, April 21, 1951), so I, failed to learn from H. Cutner's "A Short History of Sex-Worship" what are his scientific qualifications. He is in places discursive and allusive; yet he affords information of an interesting and not readily accessible subject, provides a list of reference books, whose titles often do not indicate their contents, and describes astounding survivals in some modern religions of the once widely prevalent fertility cult.

Herbert Cutner has made a useful contribution to anthropological literature.

Yours, etc.,
GUY GRIFFITHS.

131 Macquarie Street,
Sydney,
April 22, 1951.

SIDE-EFFECTS OF AMIDONE.

SIR: Three of my patients have recently reported alarming symptoms after taking amidone tablets, which are free under the *Pharmaceutical Benefits Act*. One adult, after taking only one tablet, was dizzy all day and unable to drive his car. Another adult became irrational. The third adult suffered from nausea. I notice that this drug is even made up into a cough linctus.

Yours, etc.,
F. W. SIMPSON.

105 Saint George's Terrace,
Perth.
April 23, 1951.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Bryan, C. J. D., M.B., B.S., 1950 (Univ. Adelaide), Devon Hospital, Latrobe.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, of Queensland, as duly qualified medical practitioners:

Dixon, Graham Frank, M.B., B.S. (Univ. Queensland), 61 Oriel Road, Clayfield, Brisbane.

Garozzo, Salvatore, M.B., B.S., 1951 (Univ. Queensland), Mater Hospital, South Brisbane.

McCafferty, Gerard Joseph, M.B., B.S., 1951 (Univ. Queensland), 171 Waterworks Road, Ashgrove, Brisbane.

McCracken, David Ian, M.B., B.S., 1951 (Univ. Queensland), c.o. Hospitals Board, Townsville.

McGregor-Lowndes, Victor Arnold, M.B., B.S., 1951 (Univ. Queensland), Craig Royston, Golding Street, Toowong, Brisbane.

Rosenberg, Paul, M.B., B.S., 1951 (Univ. Queensland), 128 Oxlade Drive, New Farm, Brisbane.

Farrelly, Kevin Vincent, M.B., B.S., 1951 (Univ. Queensland), Hospitals Board, Gympie.

Carr, Kenneth Allan, M.B., B.S., 1950 (Univ. Sydney), Hospitals Board, Toowoomba.

Connors, John Joseph, M.B., B.S., 1948 (Univ. Sydney), Hospitals Board, Townsville.

Kent, Merrian Winifred, M.B., B.S., 1941 (Univ. Sydney), Avalon Beach, Sydney.

The following additional qualifications have been registered:

English, Peter Bede, 113 Wickham Terrace, Brisbane, D.O.M.S., 1936, F.R.A.C.S., 1945.

Archibald, Lorna Margaret, Inchcolm, Wickham Terrace, Brisbane, D.D.M., 1949 (Univ. Sydney).

The Royal College of Obstetricians and Gynaecologists.

VICTORIAN STATE COMMITTEE.

Post-Graduate Lectures.

THE following course of post-graduate lectures of special interest to general practitioners commences on Thursday, July 5, 1951, at the College Hall, 122 Flinders Street, Melbourne, at 8.15 p.m., and will continue on alternate Thursdays until September 13, 1951: July 5, "Those Annoying Minor Disorders in Pregnancy", Bruce Anderson, M.R.C.O.G. July 19, "Rational Treatment for Vaginal Discharge", J. K. Gabriel, M.R.C.O.G. August 2, "Distressing Urinary Symptoms in the Female", Lorna Lloyd-Green, M.R.C.O.G. August 16, "Artificial Rupture of the Membranes; How, When and Why", Margaret A. Mackie, M.R.C.O.G. August 30, "What Can be Done for Menstrual Pain?", J. G. O'Donoghue, M.R.C.O.G., September 13, "How to Avoid the Retained Placenta. Management of the Third Stage", H. B. Hattam, M.R.C.O.G.

The fee for the course of six lectures is £2 2s., or 10s. 6d. for individual lectures, payable to the College at the above address, or on admission.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 21, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	15	..	..	..	..	..	15
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1(1)	1(1)	..	..	..	1	..	..	3
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	6(6)	..	2(1)	..	..	..	8
Diphtheria	20(4)	6(2)	7(3)	..	6(5)	..	..	..	39
Dysentery (Amœbic)	*	..	..	..	..	2(2)	..	..	2
Dysentery (Bacillary)	*	5(4)	9(8)	..	1(1)	..	..	..	15
Encephalitis Lethargica	..	3(2)	1	..	..	..	..	..	4
Erysipelas	*	*	*	2(1)	*	*	..	..	2
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	1	*	*	..	*	*	..	1
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	1(1)	*	*	*	*	*	1
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	2(2)	1(1)	..	..	..	..	..	3
Measles	*	*	*	31(2)	*	*	..	1	32
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	65(37)	16(2)	41(7)	14(10)	1	..	..	2	139
Psittacosis	*	*	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*c*)	*	*	*	*	1(1)	*	..	..	1
Scarlet Fever	20(10)	18(12)	7(2)	10(3)	6(4)	2(1)	..	..	63
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	65(52)	16(9)	15(5)	8(8)	11(7)	6(3)	..	..	121
Typhoid Fever(*e*)	..	..	..	..	..	..	..	..	..
Typhus (Endemic)(*f*)	..	..	..	..	2(2)	..	..	..	2
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	*	*	*	*	*	*	..
Whooping Cough	*	*	*	9(9)	*	*	..	..	9
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from the Northern Territory.

* Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Obituary.

FRANK SANDLAND HONE.

WE regret to announce the death of Dr. Frank Sandland Hone which occurred on May 9, 1951, at Adelaide.

IAN HAMILTON MACGILLIVRAY.

WE regret to announce the death of Dr. Ian Hamilton MacGillivray, which occurred on March 22, 1951, at Murwillumbah, New South Wales.

STANLEY ELDER HUMPHREYS.

WE regret to announce the death of Dr. Stanley Elder Humphreys, which occurred on May 2, 1951, at Hawthorn, Victoria.

University Intelligence.

THE UNIVERSITY OF MELBOURNE.

THE seventeenth Halford Oration will be delivered by Dr. Leslie Hurley in the Public Lecture Theatre of the University of Melbourne on June 18, 1951, at 8.15 p.m. The subject will be "The Golden Age of Medicine".

Professor E. S. J. King will deliver his inaugural lecture as Professor of Pathology in the Public Lecture Theatre of the University of Melbourne on June 25, 1951, at 8.15 p.m. The subject will be "The Story of the Melbourne School of Pathology".

Both lectures are open to all members of the medical profession.

Notice.

ROYAL PRINCE ALFRED HOSPITAL MEDICAL OFFICERS' ASSOCIATION.

THE seventeenth annual reunion of the Royal Prince Alfred Hospital Medical Officers' Association will be held at the Royal Prince Alfred Hospital, Camperdown, commencing on July 30 and terminating on Friday August 10, 1951. There will be one combined post-graduate course embracing medicine, surgery, gynæcology and obstetrics, as well as social functions.

The visit of the McIlrath Guest Professor for 1951, Mr. Gordon Murray, M.B., F.R.C.S., of Toronto General Hospital, Canada, an eminent surgeon, will coincide with the reunion and Mr. Murray will participate extensively in the clinical programme. Non-members of the Association who are graduates in medicine are eligible to attend the post-graduate course; the fee will be £5 5s. By courtesy of the Councils of Saint Andrew's College and The Women's College within the University of Sydney, accommodation will be available for a limited number of those attending the reunion, members and non-members. Early application to attend the course and for accommodation should be made to the Secretary, Royal Prince Alfred Hospital Medical Officers' Association, Camperdown, New South Wales.

A detailed syllabus of the course has already been sent to members and is available to non-members on application to the secretary.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Kotowicz, Samuel, M.B., B.S., 1950 (Univ. Queensland), 52 Sir Thomas Mitchell Road, Bondi.

Learoyd, Harry Meldrum, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Muirhead, Mortimer John Croft, M.B., B.S., 1949 (Univ. Adelaide), 14a Gloucester Street, Prospect.

Williamson, David Aubrey, M.B., B.S., 1951 (Univ. Adelaide), "Byways", Crafers.

Phillips, Colin William, M.B., B.S., 1950 (Univ. Adelaide), 3 Ettrick Avenue, Medindie.

Phillips, Pamela Rosemary, M.B., B.S., 1950 (Univ. Adelaide), 3 Ettrick Avenue, Medindie.

Diary for the Month.

MAY 22.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 23.—Victorian Branch, B.M.A.: Council Meeting.
MAY 24.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 25.—Queensland Branch, B.M.A.: Council Meeting.
MAY 31.—New South Wales Branch, B.M.A.: Branch Meeting.
MAY 31.—South Australian Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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